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Impact of Rate-Pressure Product Correction of Myocardial Blood Flow on the Prognostic Relevance in Patients with Heart Failure

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Background: Quantitative perfusion cardiac magnetic resonance (QP-CMR) enables the quantification of myocardial blood flow (MBF) and myocardial perfusion reserve (MPR), with evidence of additive prognostic benefit over traditional CMR markers [1]. For Positron Emission Tomography quantitative perfusion imaging, international guidelines recommend correcting rest MBF using the rate-pressure product (RPP = heart rate × systolic blood pressure), as elevated RPP can lead to increased rest MBF. No such guidance exists for QP-CMR. We aimed to assess the effect of RPP correction on MBF values and its prognostic value of QP-CMR in patients with heart failure (HF).

Methods: We prospectively recruited 641 patients with HF referred for clinical CMR and 184 healthy volunteers. All participants underwent CMR with standard volumes, adenosine stress/rest perfusion, and late gadolinium enhancement (LGE) (Prisma 3.0 T, Siemens). QP values were produced using an automated, in-line dual sequence method[2]. Rest and stress MBF were corrected for RPP using the formula MBF_{corr} = rest $MBF_{uncorr} \times 10{,}000/RPP$. Corrected MPR was defined as uncorrected stress MBF divided by rest MBF_{corr}. The primary endpoint was a composite of heart failure hospitalization (HFH) and all-cause mortality. Associations of corrected and uncorrected MBF/MPR with the primary endpoint were assessed using Cox regression and Kaplan–Meier curves with the log-rank test.

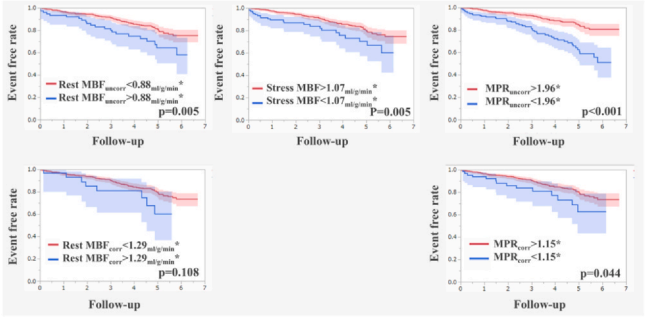
Results: RPP correction increased rest MBF and decreased MPR, but to a greater extent in healthy volunteers than in patients. During a median follow-up of 3.5 years, 101 events occurred (55 deaths and 46 HFH). Patients who reached the primary endpoint had significantly higher rest MBF_{uncorr} and lower MPR_{uncorr} than both patients who did not reach the primary endpoint and healthy volunteers. Rest MBF_{corr} and MPR_{corr} did not differ significantly between HF groups, but healthy volunteers had higher rest MBF_{corr} and MPR_{corr} than those with HF with and without events (**Table 1**). In cox regression analysis, rest MBF_{uncorr} (adjusted HR per 1 mL/g/min increase, 3.97, 95% CI 1.93–8.16, $p < 0.001$) and MPR_{uncorr} (adjusted HR per 1 unit increase, 0.74, 95% CI 0.57–0.95, $p = 0.024$) were significantly associated with the primary endpoint in both univariate and multivariate models. However, after RPP correction, these associations were no longer statistically significant (**Table 2**). Kaplan–Meier analysis revealed significant differences in event-free survival for rest MBF_{uncorr} ($p = 0.005$), stress MBF ($p = 0.005$), MPR_{uncorr} ($p < 0.001$), and MPR_{corr} ($p = 0.044$), but not for rest MBF_{corr} ($p = 0.076$) (**Figure 1**).

Conclusion: We have found uncorrected rest MBF and MPR assessed by QP-CMR to be independently associated with adverse outcomes in patients with HF. Correcting rest MBF and MPR with RPP changed MBF and MPR values and attenuated their association in a large cohort of patients with HF. Our findings support the use of uncorrected QP values in patients with HF.

Variable, median (IQR)	Heart failure patients with event	Heart failure patients without event	Healthy volunteers	p**
Rest MBF_{uncorr}, mL/g/min	0.70 (0.58–0.87) *†	0.61 (0.52–0.73)*	0.57 (0.48–0.68)	< **0.001**
Rest MBF_{corr}, mL/g/min	0.77 (0.64–0.97) *	0.73 (0.61–0.92)*	0.92 (0.78–1.06)	< **0.001**
Rest RPP, mmHg	9006 (7245–10952)*†	8255 (6964–9773) *	6336 (5518–7403)	< **0.001**
Stress MBF, mL/g/min	1.48 (1.12–1.93) *	1.57 (1.25–1.95)*	2.16 (1.81–2.49)	< **0.001**
Stress RPP, mmHg	9711 (8345–11512)*	10147 (8362–12081)*	8570 (7526–10113)	< **0.001**

MPR_{uncorr}	2.04 (1.43–2.72)*†	2.53 (2.03–3.14)*	3.63 (3.08–4.40)	< **0.001**
MPR_{corr}	1.93 (1.38–2.42)*	2.07 (1.58–2.69)*	2.31 (2.00–2.79)	< **0.001**

	Primary outcome: Composite of Death or HFH			
Predictor	Unadjusted HR (95% CI)	P value	Adjusted HR* (95% CI)	P value
Rest MBF_{uncorr}	3.43 (1.52–6.84)	0.001	3.97 (1.93–8.16)	< 0.001
Rest MBF_{corr}	1.13 (0.52–2.31)	0.749	1.29 (0.63–2.62)	0.489
Stress MBF	0.68 (0.47–0.99)	0.045	0.90 (0.60–1.33)	0.605
MPR_{uncorr}	0.65 (0.51–0.84)	0.001	0.74 (0.57–0.95)	0.024
MPR_{corr}	0.75 (0.58–0.96)	0.028	0.84 (0.65–1.07)	0.179



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Epicardial adiposity is associated with impaired cardiac energetics in immune-mediated inflammatory diseases.

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Background: Immune-mediated inflammatory diseases (IMIDs) are associated with increased risks of cardiovascular disease (CVD) and mortality mainly driven by systemic inflammation. Odds ratios for myocardial infarction are >2 in most IMIDs. Epicardial, subcutaneous and visceral adiposity are also linked to increased cardiovascular inflammation and disease. Whether adiposity impacts myocardial structure and function in patients with IMIDs is unknown.

Methods: Twenty-one patients with systemic sclerosis (SSc), 14 with positive anti-cyclic citrullinated peptide antibody (CCP + ve) at high risk of developing rheumatoid arthritis (RA), 14 with established RA, and 18 age, sex, and body mass index (BMI) matched healthy volunteers (HV) underwent Phosphorus-31 magnetic resonance spectroscopy (31P-MRS) and cardiac magnetic resonance (CMR) on a Siemens 3 Tesla PRISMA system (Erlangen, Germany).

In addition to volumes, a fat/water separated multi-echo GRE sequence was acquired, and fat images manually contoured in cvi42 (Calgary, Canada). Areas of epicardial adiposity tissue (EAT) were averaged over 10 long-axis slices from the level of the great vessels to the true cardiac apex. Areas of abdominal subcutaneous (SAT) and visceral (VAT) adiposity tissue were averaged over 5 contiguous axial slices centred at L2-L3 intervertebral disc level.

31P-MRS data were acquired with non-gated 3D acquisition weighted chemical shift imaging (CSI). Post processing analysis was performed with custom Matlab software (Mathworks, Natick, Mass).

Results: Bi-ventricular size, function, left ventricular (LV) mass index, left atrial (LA) volume and function were normal across all groups (Table-1).

SAT (cm2) was significantly increased in RA (RA: 272 [194-322], HV: 154 [116-221], p = 0.0015). VAT (cm2) was significantly increased in anti-CCP + ve patients (CCP: 203 [176-338], HV: 127 [75-185], p = 0.0248). EAT (cm2) was significantly increased in SSc (23 [17-29]), anti-CCP (40 [29-46]), and chronic RA (33 [22-42]) versus HV (19 [15-31], p = 0.0008).

All three patient groups had reduced cardiac energetics with significantly reduced phosphocreatine-to-adenosine triphosphate (PCr/ATP) ratio (SSc: 1.8 [1.7-2.2], CCP: 1.8 [1.5-2], SSc: 1.8 [1.6-2.2], HV: 2.3 [2.2-2.5], p = 0.0126).

EAT correlated negatively with PCr/ATP (r -0.33, p = 0.0178) and global longitudinal strain (r -0.38, p = 0.0413). Both EAT and VAT correlated positively with male sex, age, BMI, body surface area (BSA), and HBA1c (all p < 0.05). VAT correlated with worse lipid profile (p < 0.05). SAT correlated positively with male sex, BMI, and BSA (all p < 0.05) without impact on cardiac functions.

Conclusion: We report for the first time that epicardial adiposity tissue is associated with subclinical reduction in myocardial energetics and global longitudinal strain in patients with IMIDs and CCP positive patients at-risk of RA. This underlines the importance of CVD risk factor modulation in this patient group, especially when EAT is increased.

Table 1 Clinical, CMR and 31 P MRS findings

	HV (n = 18)	SSc (n = 21)	CCP + ve (n = 14)	RA (n = 14)	P value
Age, y	45 [31-63]	53 [38-63]	55 [51-66]	62 [47-67]	0.0707
Female, n (%)	14 (78)	13 (62)	8 (57)	11 (79)	0.4308
BMI, kg/m²	27 ± 5	25.5 ± 5.1	29.3 ± 4.3	27.3 ± 2.9	0.1157
Body surface area, m²	1.9 ± 0.2	1.8 ± 0.2	2.0 ± 0.2	1.9 ± 0.2	0.2629
Systolic BP, mmHg	124 [112-136]	114 [100-130]	125 [122-138]	127 [113-139]	0.2588
NT- Pro BNP, ng/L	61 [36-103]	86 [57-183]	77 [35-105]	105 [66-145]	0.3052
Troponin I, ng/L	3 [3-6]	4 [3-8]	3 [3-8]	5 [3-8]	0.7944
HbA1c, mmol/mol	35 [34-39]	34 [33-38]	38 [37-42]	38 [35-42]	**0.0195**
Total cholesterol, mmol/L	5.1 [4.3-6.3]	5.0 [4-5.7]	5.3 [4.3-6.3]	4.8 [4.3-5.7]	0.7247
HDL cholesterol, mmol/L	1.8 [1.3-2.1]	1.4 [1.1-1.9]	1.3 [1.1-1.7]	1.7 [1.3-2.1]	0.1250