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Unique role of admission hyperglycemia on myocardial infarction size and area at risk following an acute ST-elevation myocardial infarction

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Abstract

Background: Hyperglycemia can adversely affect patients with acute ST-elevation myocardial infarction (STEMI) in both diabetic and non-diabetic patients. The majority of the studies had investigated the impact of admission hyperglycemia (AH) on cardiovascular morbidity and mortality while, in ours, we entailed its impact on final infarction size (FIS) and more interestingly, on the area at risk (AAR), both were estimated by cardiac magnetic resonance (CMR) imaging.

Results: AH showed significant positive correlations to FIS and AAR. Moreover, AH group had higher summation of ST segment elevation (sum STE), more maximum ST segment elevation (max STE), higher echocardiographic wall motion score index (WMSI), higher CMR estimated WMSI, and lower segmental ejection fraction (EF). Multivariate analysis showed that AH was independently associated with increased FIS.

Conclusion: Current study showed an association between AH and large FIS in STEMI patients.

Keywords: Admission hyperglycemia, ST-elevation myocardial infarction, Infarction size

Background

Accounting for more than 15 million deaths annually, cardiovascular diseases are the leading causes of death worldwide [1]. In the last decade, the mortality rates after ST-elevation myocardial infarction (STEMI) showed an obvious decline which is explained by the expanded use of primary percutaneous coronary intervention (PPCI), fibrinolytic therapies, and updated antithrombotics [1]. But, in Egypt, as well as low- and middle-income countries, mortality and cardiovascular events are still relatively high with the comparable burden with that of infectious diseases and other non-communicable diseases, despite improvement in therapeutic and interventional therapy [1].

Admission hyperglycemia (AH) has been determined to inversely affect short- and long-term prognosis in patients with STEMI regardless of their diabetic state [2].

Single-photon emission computed tomography (SPECT) is the traditional imaging tool utilized to measure FIS, but it has a limited spatial resolution, and carry the risk of radiation [3]. CMR is a relatively new cardiac imaging modality that can assess changes in cardiac contractile function, metabolic activity, and membrane integrity. Furthermore, CMR has a high spatial and temporal resolution which facilitates accurate assessment of cardiovascular structures. By the addition of late gadolinium enhancement (LGE) with CMR, transmural extension and scar tissues can be estimated [4].

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A few data about the sequelae of AH on FIS after acute STEMI patients are present and nearly no available data about the relation between AH and area at risk, and the objective of the current study was to investigate the relationship between admission blood glucose and myocardial FIS and AAR after reperfusion in STEMI patients by using CMR.

Methods

Study population

Forty-three patients who were presented with their first acute STEMI and underwent successful reperfusion, in a period less than 12 h from onset of symptoms, were included. They were selected from the Critical Care Unit, Internal Medicine Department, from April 2018 to May 2019. Patients were informed about the research and signed informed consent. Acute STEMI is diagnosed when there is chest pain lasting ≥ 30 min; ST-segment elevation defined according to the European Society of Cardiology/ACCF/AHA as a new ST elevation at the J point affecting at least 2 contiguous leads of ≥ 2 mm in males or ≥ 1.5 mm in females in leads V2–V3 and/or of ≥ 1 mm in remaining contiguous chest or limb leads [5]. Successful reperfusion determined by resolution of ST-segment ≥ 50% 1 h after thrombolysis, which can be classified into partial resolution [50–70%] or complete resolution [> 70%].

Patients ≥ 85 years old, with life-limiting non-cardiac disease, prior STEMI, left bundle branch block (LBBB), any contraindications to MRI, and an estimated glomerular filtration rate < 30 ml/min/1.73 m^2, who were hemodynamically unstable to be transferred to CMR laboratory were excluded.

According to AH they were divided into 2 groups (patients with AH = 21 and patients without AH = 22). AH was defined as the presence of random plasma glucose > 140 mg/dl in non-diabetic patients within 2 h of admission [5]. Plasma glucose levels (PG) were assessed every 2 h; mean PG levels were calculated at days 1, 2, and 3 and just before CMR.

Data Collection

Demographic data including age, gender, body mass index, history of hypertension, diabetes mellitus, dyslipidemia, smoking, duration of chest pain, Killip class, and electrocardiogram (ECG) data were collected. Venous blood samples were taken on admission to measure glucose level and glycated hemoglobin (HbA1c). Low density lipoprotein (LDL) was assessed from fasting venous blood samples taken on the first morning after admission. Peak plasma values of creatine kinase (CK) and CK-MB were also evaluated. Baseline serum creatinine clearance was also evaluated by the Cockcroft-Gault formula [6].

CMR protocol

Cardiac MRI was performed 4 to 6 days after acute STEMI with a 1.5-Tesla scanner (Philips, Achieva) using a 6-channel body array coil. Left ventricular (LV) long-axis scout images were taken to adjust the LV short-axis imaging planes [7]. Short-axis slices (thickness of 5 mm with a slice gap of 10 mm) were defined from the base to the apex of the heart. Imaging of the heart was performed at the end-systolic time to estimate left ventricular end-systolic volume (LVESV) and at the end-diastolic time to estimate left ventricular end-diastolic volume (LVEDV). Steady-state free precession sequences were used for cine imaging; a dark-blood T2-weighted short-tau inversion-recovery turbo-spin echo sequence (STIR) was applied to determine AAR (with edema), and a segmented phase-sensitive inversion-recovery (PSIR) steady-state free precession sequence was used for late enhancement imaging 10 min after intravenous injection of 0.15 mmol/kg of gadolinium diethylenetriaminepentaacetic acid (Gd-DTPA) (Magnevist; Bayer Schering, Berlin, Germany). All images were ECG-triggered and acquired during breath-holds. CMR studies were analyzed then offline (Philips Medical Systems, Nederland B.V).

CMR data analysis

Data were analyzed by using a 17-segment model. From short-axis views, both LVEDV and LVESV were measured. Left ventricular ejection fraction (LVEF) was calculated as LVEF = (EDV − ESV)/EDV.

For each patient, AAR was recognized as hyper-intense zones on T2-weighted images; left ventricular endo- and epicardial borders had been delineated; then, reference normal area was drawn. AAR was defined as the percentage of LV volume delineated by the hyper-intense zone on T2-STIR images with signal intensity was > 2 standard deviations above the mean signal obtained in the distant non-infarcted myocardium and was expressed as a percentage of normal myocardium. FIS was measured on PSIR sequences by delineating epicardial and endocardial borders; the papillary muscle was considered as a part of the cavity of the left ventricle; then, a myocardial area was regarded as hyper-intense whenever the signal intensity was > 2 standard deviations above the mean signal intensity in the non-infarcted myocardium; then, the FIS was expressed as a percentage of normal non-infarcted myocardium (Fig. 1).

Statistical analysis

IBM-SPSS 21 was used for the analysis of data. The normality was tested by the Kolmogorov–Smirnov test. The continuous variables were presented as the means ± standard deviation (SD), and categorical variables were expressed in the form of percentages. The difference in

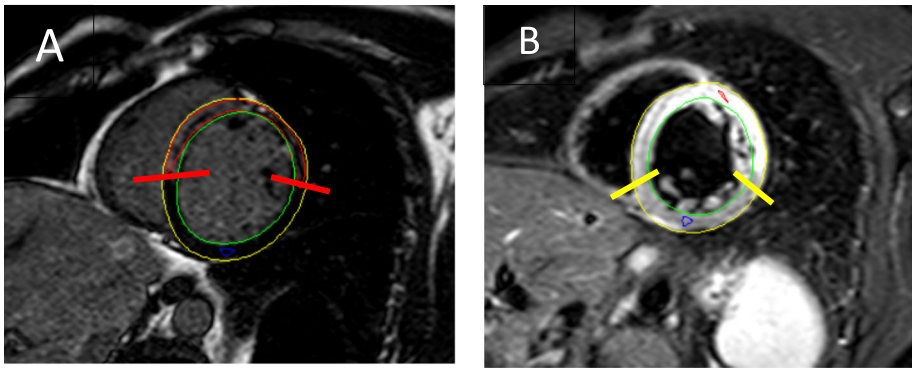

Fig. 1 Cardiac MRI at 1 week in a patient with a large anterior myocardial infarction. **a** Late enhancement imaging allowed for the definition of infarct size (hyper-enhanced myocardium, between red arrows). **B** T2-weighted sequence demonstrated a large area at risk (with edema, between yellow arrows)

frequencies among different groups was compared by Chi-square which was used to test differences in categorical data between groups, while Student *t* test and Mann-Whitney *U* test were used to test differences in means in continuous variables between groups (parametric and non-parametric). Spearman rank correlation coefficient was calculated for univariate correlations between echo and CMR parameters. A *p* value of less than 0.05 was considered significant. Multivariate linear regression analysis was performed to test the independent predictors of FIS.

Results

Clinical and echocardiographic characteristics

The data showed that male is the predominate gender (86.4% of the euglycemic group and 81% of AH group) with insignificant differences between both groups as regards mean age (*p* value = 0.487) and gender (*p* value = 0.473). Patients with AH had a significantly longer chest pain duration, higher heart rate on discharge, higher percent of anterior MI, higher summation of ST elevations (sum STE) on admission and maximum ST-segment elevations (max STE) on admission, and higher baseline echocardiography-derived classic WMSI (Table 1).

In comparing PG levels between both groups, there was insignificant difference in all days before CMR performance; only they were significantly different in admission plasma glucose levels (Table 2).

CMR findings

Although there were no differences in LVEDV, LVESV, and EF between the two groups, the AH group had significantly higher classic WMSI, larger FIS, and AAR (Table 3).

Correlation analysis

Figure 2 shows that there was a significant positive correlation between admission plasma glucose (APG), FIS, and AAR in patients with STEMI.

Regression analysis

The multivariate linear regression analysis of the significant factors affecting myocardial CMR LGE infarction size. After adjusting for age, the final linear regression model contained six predictors: chest pain duration, APG, peak CK-MB, sum ST, max ST, and WMSI estimated by CMR (Table 4).

Discussion

Despite improvements in medical and interventional therapies in the last 40 years, acute STEMI is still a leading cause of mortality and morbidity. Many studies were done to determine possible factors that may increase poor outcomes in STEMI patients, mostly following myocardial remodeling. Admission hyperglycemia is a unique factor deserving a great interest to investigate its role in the outcomes of STEMI patients, with special emphasis on its relation to FIS and AAR.

Roles of hyperglycemia in patients with STEMI have many points of interest, first, chronic hyperglycemia which present even before AMI in diabetic patients, increased myocardial susceptibility to infarction in DM patients was indicated by two clinical studies, which detected significantly larger infarct size measured by SPECT in diabetic than non-diabetic patients [8]. This can be explained by impaired collateral recruitment, microcirculatory abnormalities, and underuse of evidence-based therapies [9]; second, hyperglycemia during hospital stay, and lastly and more interestingly, AH which is a unique predictor studied by our research and attracted more attention to the need of more strict control of admission plasma glucose.

Egypt has nearly 100 million people who have special demographic and social characteristics which increase the possibility of the stress-induced conditions and its health-associated problems; some of these characteristics

Table 1 Patients' clinical and echocardiographic characteristics

	Euglycemic, *n* = 22	Admission hyperglycemia, *n* = 21	*p* value
Age/years (mean ± SD)	56.49 ± 11.4	58.95 ± 11.3	0.487*
Sex (males%)	19 (86.4%)	17 (81%)	0.473**
Hypertension *n* (%)	12 (54.5%)	9 (42.9%)	0.443**
Smoking *n* (%)	15 (68.2%)	11 (52.4%)	0.289**
Chest pain duration/h (mean ± SD)	6.36 ± 3.2	9.81 ± 5.2	**0.019*** **
BMI (kg/m2)	26.64 ± 4.8	25.52 ± 4.7	0.448*
Heart rate (beat/min)	84.09 ± 14.0	92.05 ± 11.2	0.050*
SBP (mm/Hg)	118.46 ± 21.7	122.38 ± 23.2	0.587*
DBP (mm/Hg)	75.91 ± 11.2	77.14 ± 15.1	0.767*
Killip score			0.951*
Class I	19 (86.4%)	18 (85.7%)	
Class II	3 (13.6%)	3 (14.3%)	
Anterior *n* (%)	7 (31.8%)	13 (61.9%)	**0.048****
Admission sum of STE/mm (mean ± SD)	11.09 ± 9.8	18.14 ± 7.9	**0.002*****
Admission max STE/mm (mean ± SD)	3.59 ± 2.3	5.10 ± 1.8	**0.003*****
Resolution of STE *n* (%)			0.625**
≥ 50–70	10 (45.5%)	8 (38.1%)	
≥ 70	12 (54.5%)	13 (61.9%)	
LDL (mg/dl) (mean ± SD)	108.41 ± 35.5	106.38 ± 28.6	0.874***
S. creatinine (mg/dl) (mean ± SD)	0.95 ± 0.2	1.01 ± 0.2	0.259***
Initial CK-MB (ng/ml) (mean ± SD)	57.14 ± 36.9	64.76 ± 42.5	0.381***
Peak CK-MB (ng/ml) (mean ± SD)	245.23 ± 184.3	306.38 ± 174.2	0.162***
HBA1c	5.11 ± 0.9	5.50 ± 0.9	0.087***
Classic WMSI (mean ± SD)	1.38 ± 0.3	1.52 ± 0.2	**0.042*****
LVESV (ml) (mean ± SD)	41.71 ± 13.7	40.96 ± 12.7	0.356***
LVEDV (ml) (mean ± SD)	81.95 ± 18.5	75.60 ± 15.9	0.789***
EF % (mean ± SD)	54.05 ± 11.5	50.81 ± 12.0	0.462***

p value ≤ 0.05 is significant
BMI body mass index, *CK* creatinine kinase, *DBP* diastolic blood pressure, *EF* ejection fraction, *HBA1c* glycated hemoglobin, *LDL* low density lipoprotein, *LVEDV* left ventricular end diastolic volume, *LVESV* left ventricular end systolic volume, *Max STE* maximum ST-segment elevation, *SBP* systolic blood pressure, *SD* standard deviation, *Sum of STE* summation of ST-segment elevations, *WMSI* wall motion score index
*Independent *t* test was used to compare the mean difference between groups
**Chi-square test was used to compare the proportion differences
***Mann-Whitney *U* test was used to compare the median difference between groups

Table 2 Comparison between the stress hyperglycemia group and the euglycemic group as regards plasma glucose levels in all days before cardiac magnetic resonance

	Euglycemic group (no. 22)	Stress hyperglycemic group (no. 21)	*p* value
Mean APG	223.45	11.33	< 0.0001
Day 1 mean PG level (mg/dl)	142.64	121.44	0.09
Day 2 mean PG level (mg/dl)	122.64	11.38	0.06
Day 3 mean PG level (mg/dl)	115.36	114.28	0.17
Mean PG level before just CMR (mg/dl)	113.14	116.23	0.10

p value < 0.05 is significant
APG admission plasma glucose, *CMR* cardiac magnetic resonance, *PG* plasma glucose
*Independent *t* test was used to compare the mean difference between groups

Table 3 Cardiovascular magnetic resonance imaging findings

	Euglycemic, $n = 22$	Admission hyperglycemia, $n = 21$	p value*
LVESV (ml)	75.42 ± 21.2	81.39 ± 26.9	0.360
LVEDV (ml)	136.71 ± 22.2	140.89 ± 24.1	0.679
EF %	51.09 ± 12.8	49.45 ± 13.1	0.590
Classic WMSI	1.39 ± 0.3	1.56 ± 0.2	**0.021**
FIS %	19.73 ± 9.5	28.05 ± 13.1	**0.022**
AAR %	31.59 ± 13.4	39.62 ± 16.6	0.135

p value < 0.05 is significant. Data were expressed in mean ± SD
AAR area at risk, *EF* ejection fraction, *FIS* final infarct size, *LVEDV* left ventricular end diastolic volume, *LVESV* left ventricular systolic volume, *MVO* microvascular obstruction, *SD* standard deviation, *WMSI* wall motion score index
*Mann-Whitney *U* test was used to compare the median difference between groups

may be shared with other developing nations. Previous Egyptian studies confirmed that elevated admission glucose level is a strong predictor of short-term adverse outcomes in patients with AMI but no previous studies in our country regarding the impact of AH on AAR, FIS, or SI which is the major concept of our research [10, 11].

An Indian study had addressed the association of AHG and increased myocardial damage evidenced by cardiac biomarkers and echocardiographic derived data but without using CMR modality. In that study, increased admission plasma glucose was associated with lower LVEF, higher WMSI, and higher CKMB in STEMI patients, all in line with our results except for LVEF which had an insignificant association with AHG in our studied population [12].

One major finding in our study is that AAR was positively correlated with APG; this is explained by the fact that acute hyperglycemia may increase the inflammatory response during STEMI and could thus influence microvascular permeability and edema [13]. To the limit of our knowledge, few studies investigated the impact of stress hyperglycemia on AAR [14]. Another study found no association between AHG and FIS [15].

Moreover, admission plasma glucose associated with larger FIS estimated by CMR and assessed by delayed enhancement (DE) score in acute STEMI patients [16]. However, the mechanism by which admission hyperglycemia adversely affect myocardial FIS is not well understood. But it was proved that admission hyperglycemia strongly predicted the reduction of epicardial flow in the infarct-related vessel before reperfusion therapy in patients with STEMI [17]. Moreover, admission hyperglycemia may be a consequence of large infarction size rather than the cause; as infarction increases in size, more catecholamines are secreted, which in turn affect fatty acids and glucose secretion [18].

Several studies have investigated the clinical impact of LGE on patient outcome after AMI. However, few studies have evaluated clinical factors or laboratory parameters that may be predictive for the development of LGE. Identification of such predictors for the development of LGE post-AMI may facilitate early risk stratification, even before cardiac imaging. In line with our study, a previous one supported that delayed presentation was predictor for larger infarction size [19]. In contradiction to our results, a previous one found no significant relationship between the time to PCI and infarction size [20]. Supporting to our data, a previous study revealed that high CK-MB values were independent predictors of LGE [21]. Similarly, Klug et al. observed a correlation between CK value and infarct size in reperfused STEMI [22].

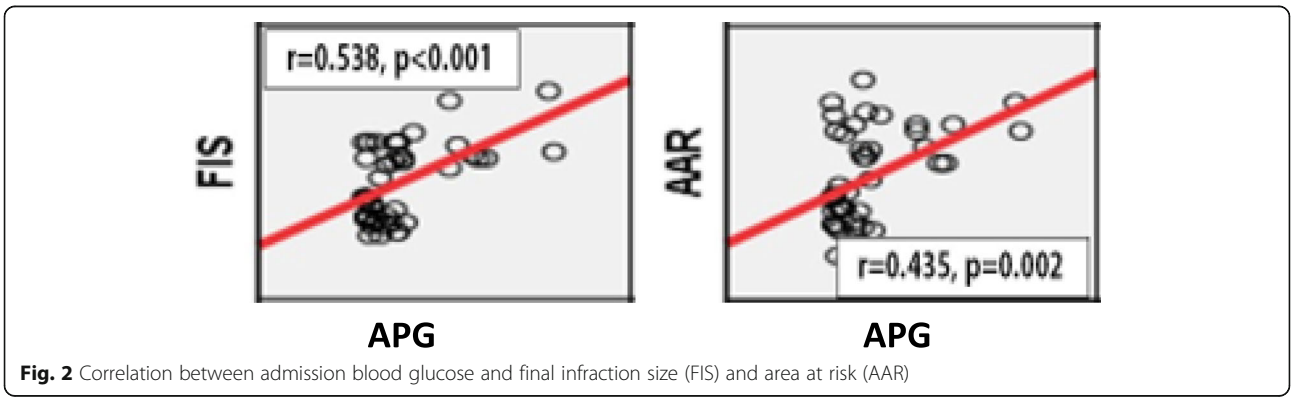

Fig. 2 Correlation between admission blood glucose and final infraction size (FIS) and area at risk (AAR)

Table 4 The multivariate linear regression analysis of the factors affecting FIS

	Estimate	SE	*t* stat	*p* value
Intercept	**5.87 (1.05–14.18)**	**1.14**	**4.87**	**= 0.011**
Age/years	0.02 (− 0.34–0.37)	0.23	0.09	= 0.928
Heart rate (beat/min)	0.13 (− 0.15–0.41)	0.12	0.96	= 0.343
Chest pain duration (h)	**1.04 (0.28–1.80)**	**0.41**	**2.75**	**= 0.009**
APG (mg/dl)	**0.08 (0.04–0.11)**	**0.02**	**4.09**	**< 0.001**
Peak CK-MB (ng/ml)	**0.16 (0.08–0.24)**	**0.04**	**3.99**	**< 0.001**
Sum ST elevation	**0.71 (0.38–1.04)**	**0.16**	**4.35**	**< 0.001**
Max ST elevation	**2.57 (1.03–4.10)**	**0.76**	**3.38**	**= 0.002**
Echo WMSI	-1.73 (− 5.29–1.95)	0.91	0.84	= 0.624
CMR WMSI	**1.89 (1.01–3.51)**	**0.18**	**4.62**	**= 0.005**
Infarction site (anterior)	4.07 (− 2.84–10.98)	3.40	1.29	= 0.240

p value < 0.05 is significant
APG admission plasma glucose, *CK* creatinine kinase, *CMR* cardiac magnetic resonance, *Max ST elevation* maximum ST elevations, *Sum ST elevation* summation of ST elevations, *WMSI* wall motion score index

In this small study, we tried to start highlighting the ability of CMR-derived prognosticators to detect the impact of AHG on FIS and AAR in this cohort of patients in Egypt without neglecting less accurate but more available and less costly ECG and echocardiographic data.

Study limitations

Our study did not include any data regarding details of insulin therapy given to the patients with admission hyperglycemia which may influence the FIS and AAR. So, it is recommended to investigate the role of glucose-lowering therapy in hyperglycemic patients on FIS. Our study interpretation should be applied to low- and moderate-risk STEMI patients as high-risk STEMI (e.g., higher Killip class or cardiogenic shock) patients are excluded because they were not able to continue the procedure. Financial problems and lack of funding support restricted the sample size which decreases the validity of the studied variables.

Conclusions

Admission plasma glucose positively correlates with CMR estimated FIS and AAR in successfully reperfused STEMI.

Abbreviations

AAR: Area at risk; AH: Admission hyperglycemia; APG: Admission plasma glucose; CK: Creatine kinase; CMR: Cardiac magnetic resonance; DE: Delayed enhancement; ECG: Electrocardiogram; EF: Ejection fraction; FIS: Final infarction size; Gd-DTPA: Gadolinium diethylenetriaminepentaacetic acid; HbA1c: Glycated hemoglobin; LBBB: Left bundle branch block; LDL: Low density lipoprotein; LGE: Late gadolinium enhancement; LV: Left ventricular; LVEDV: Left ventricular end-diastolic volume; LVESV: Left ventricular end-systolic volume; Max STE: Maximum ST segment elevation; PPCI: Primary percutaneous coronary intervention; PSIR: Phase sensitive inversion recovery; SD: Standard deviation; SPECT: Single-photon emission computed tomography; STEMI: ST-elevation myocardial infarction; STIR: Short-tau inversion-recovery turbo-spin echo sequence; Sum STE: Summation of ST segment elevation; WMSI: Wall motion score index

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Authors' contributions

1. T.M: the acquisition and analysis of data, approved the submitted version, and agreed both to be personally accountable for the author's own contributions and to ensure that questions related to the accuracy or integrity of any part of the work are appropriately investigated, resolved, and the resolution documented in the literature. 2. M.A: put the concept of the research, approved the submitted version, and agreed both to be personally accountable for the author's own contributions and to ensure that questions related to the accuracy or integrity of any part of the work are appropriately investigated, resolved, and the resolution documented in the literature. 3. R.A: put the design of the research, approved the submitted version, and agreed both to be personally accountable for the author's own contributions and to ensure that questions related to the accuracy or integrity of any part of the work are appropriately investigated, resolved, and the resolution documented in the literature. 4. H.I: revision of the work, approved the submitted version, and agreed both to be personally accountable for the author's own contributions and to ensure that questions related to the accuracy or integrity of any part of the work are appropriately investigated, resolved, and the resolution documented in the literature. 5. D.H: interpretation of data, approved the submitted version, and agreed both to be personally accountable for the author's own contributions and to ensure that questions related to the accuracy or integrity of any part of the work are appropriately investigated, resolved, and the resolution documented in the literature. All authors have read and approved the manuscript.

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Availability of data and materials

- Patients are recruited from those who were admitted to Critical Care Unit, Internal Medicine Department, Assiut University.
- CMR was performed in Radiology Department, Assiut University.
- Gadolinium used in CMR was on our expense as there was no any funding support.

Ethics approval and consent to participate

"The Committee of Medical Ethics" of Faculty of Medicine, Assiut University, had approved the study with reference no. 17200407.
Written consents were obtained from all patients.

Consent for publication

Not applicable.
No identifying images nor personal data were included in the manuscript.

Competing interests

None.

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