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ORIGINAL RESEARCH

Intramyocardial Hemorrhage in Patients with Acute Myocardial Infarction Without Reperfusion Therapy: A Prospective Study

Muzheng Li[1], Zhijian Wu[2], Ilyas Tudahun[3], Kun Zhang[1]

[1]Department of Cardiology, The Seventh Affiliated Hospital of Sun Yat-Sen University, Shenzhen, 518107, People's Republic of China; [2]Department of Pulmonary and Critical Care Medicine, The Second Xiangya Hospital, Central South University, Changsha, Hunan, 410011, People's Republic of China; [3]Department of Cardiology, Changsha Third People's Hospital, Changsha, Hunan, 410008, People's Republic of China

Correspondence: Kun Zhang, Department of Cardiology, The Seventh Affiliated Hospital of Sun Yat-sen University, Shenzhen, 518107, People's Republic of China, Email zhangkun@sysush.com

Background and Aims: IMH commonly presents in STEMI patients receiving reperfusion therapy and is considered as an ischemic reperfusion injury. However, it is unclear whether IMH occurs in AMI patients without reperfusion therapy.

Methods and Results: We prospectively enrolled 40 patients with STEMI and 41 patients with NSTEMI admitted to the CCU of the Second Xiangya Hospital of Central South University from April 2020 to November 2021, all of whom did not receive reperfusion therapy. In the STEMI group, 16 patients were detected with IMH by CMR. However, in the NSTEMI group, only 3 patients were detected. The incidence of IMH was significantly higher in patients with STEMI than NSTEMI (16/40 vs 3/41, $P < 0.001$). Among patients with STEMI, the incidence of IMH was not significantly different between patients who underwent primary percutaneous coronary intervention and those who did not (16/40 vs 27/65, $P = 0.876$). Patients in the spontaneous reperfusion group had a higher incidence of IMH than patients in the non-spontaneous reperfusion group (11/23 vs 5/17, $P = 0.240$). Similarly, in patients with STEMI who did not receive reperfusion therapy, the incidence of MACE was higher in the IMH-present group than in the IMH-absent group (5/16 vs 2/24, $P = 0.063$).

Conclusion: The incidence of IMH is comparable in patients with STEMI with or without reperfusion therapy, but considerably higher than that in NSTEMI patients. Patients with STEMI can present with IMH even when infarct-related vessel flow is not restored.

Keywords: myocardial infarction, intramyocardial hemorrhage, outcome, reperfusion therapy

Introduction

Reperfusion therapy is the most essential and critical treatment for acute myocardial infarction (AMI), and timely opening of the occluded vessel, whether using thrombolysis or percutaneous coronary intervention (PCI), has been proven to significantly reduce angina or heart failure symptoms and improve outcomes.[1] However, reperfusion therapy also frequently brings about side effects, such as ischemia-reperfusion (IR) injury.[2] IR injury in addition to involving cardiomyocytes, vascular endothelial cells are similarly affected.[3] Reperfusion vessel injury can cause coronary microcirculatory disorders, leading to the no-reflow phenomenon after PCI, which is considered to be closely related to microvascular obstruction (MVO) and intramyocardial hemorrhage (IMH).[4,5] IMH refers to microvascular injury within the infarct area that leads to extravasation of blood components, such as erythrocytes to the myocardial interstitium, which can be detected by cardiac magnetic resonance (CMR).[6] Increasing evidence suggests that patients with AMI complicated by IMH have a relatively poor prognosis after discharge. IMH can increase the incidence of major adverse cardiovascular events (MACE) such as heart failure and sudden death in AMI patients.[7] In addition, previous studies have revealed that the incidence of IMH in patients with acute ST segment elevation myocardial infarction (STEMI) is extremely common.[8–10] A meta-analysis study that reviewed 9 retrospective studies showed that the incidence of IMH in

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STEMI patients after PCI was approximately 35%. This is an important phenomenon that needs our attention in the field of AMI.

However, there are still some limitations in the current knowledge in the field of IMH. First of all, most of the studies related to the incidence and prognosis of IMH were conducted in STEMI patients who successfully opened their culprit vessels after PCI.[11] While another condition, probably with a larger number of patients, it is unknown whether IMH occurs in patients with non-ST segment elevation myocardial infarction (NSTEMI) and whether IMH affects outcomes in these patients. Secondly, most studies believe that IMH is related to IR injury, and it is unclear whether IMH occurs in AMI patients who do not receive reperfusion therapy.

Therefore, we designed a single-center, prospective study to investigate the incidence and clinical characteristics of IMH in patients with two types of AMI who did not receive reperfusion therapy.

Methods

Patients

We prospectively enrolled consecutive newly diagnosed AMI (STEMI and NSTEMI) cases admitted to the Coronary Care Units (CCU) of the Second Xiangya Hospital of Central South University from April 2020 to November 2021, all of whom did not receive reperfusion therapy. The diagnostic criteria are according to the fourth edition of the global definition of myocardial infarction (2018).[7] Patients with clear prior reperfusion therapy (thrombolysis, PCI, or CABG) for ischemia were excluded (For specific inclusion and exclusion criteria, see Supplementary Material 1). The study protocol conformed to the ethical guidelines of the Declaration of Helsinki as reflected by prior approval from the human research committee of the Second Xiangya Hospital of Central South University, the ethics approval number is (Y2021453-2). Written informed consent was obtained from patients while the patient was in a clinically stable, non-congested condition or their family members who can give informed consent on behalf of patients after they were informed about the objectives and procedures of the study. Their right to refuse participation at any time they want was assured. For this purpose, a one-page consent letter was attached as a cover page of each questionnaire stating the general objective of the study and issues of confidentiality that were discussed by the data collectors before proceeding to the data collection.

Data Collection

Medical records are from the inpatient and emergency medical system. Data including demographic characteristics, comorbidities, laboratory testing results, electrocardiographic (ECG), PCI record, echocardiographic findings and treatment were obtained. Follow-up started at the time of diagnosis of AMI. The primary clinical endpoint [major adverse cardiac events (MACE)] was defined as a composite of cardiac death, reinfarction and the occurrence of new heart failure (HF) after hospital discharge for the index event. To avoid double counting of patients with more than one event, each patient contributed only once to the MACE endpoint (death > reinfarction > HF). Data comes from medical records or telephone interviews with patients or relatives by two trained doctors. The final date of follow-up was September 16, 2022. Survival time (months) was measured as the duration between the first day of hospitalization when the patient was diagnosed with STEMI to the date of MACE.

CMR Protocol

Patients underwent CMR on 3.0 T scanners (MAGNETOM Skyra, Siemens Healthcare, Erlangen, Germany) with an 18-channel body coil combined with the spine coil after admission to determine whether IMH occurs. All CMR image analyses were performed using CVI42 (Circle Cardiovascular Imaging Inc) by 2 radiologists with more than 3 years of CMR experience in consensus. IMH was assessed by T2 or T2 mapping quantification using a breath-hold, cardiac gated gradient echo sequence with eight echoes obtained in three matching short-axis slices before administration of the contrast agent. IMH was defined as a region of hypointense core within the infarcted area with reduction of T2 signal intensities ≤ 20 ms (Figure 1 presents 2 representative clinical figures). The infarct volume and MVO were measured with left ventricular short axis delayed gadolinium enhanced (LGE) images.

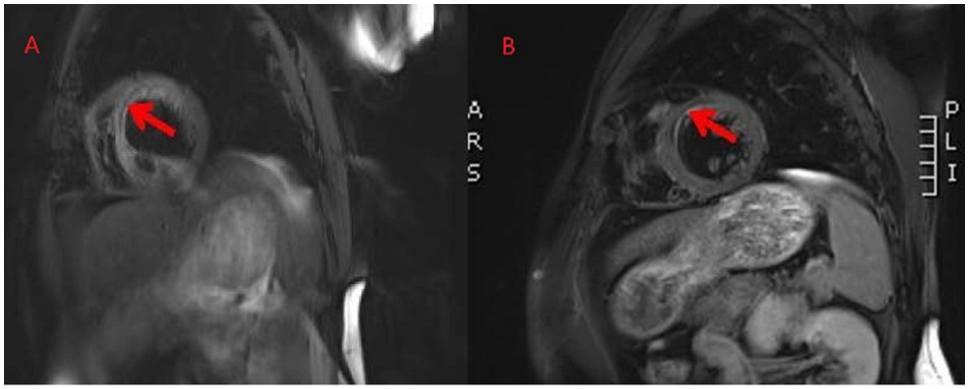

Figure 1 (**A**) A 58 year old male patient with STEMI was transferred to the emergency department after PCI. CMR on the third day showed that IMH presented in the interventricular septum and the infarcted core area of the left ventricular anterior wall; (**B**) A 46 year old male STEMI patient visited our hospital due to chest tightness for 5 days, and CMR revealed IMH in the interventricular septum and infarct core area of the anterior wall of the left ventricle.

Statistical Analysis

Normal distribution parameters are expressed as mean ± standard deviation (SD), while non-normal distribution parameters are expressed as median (Q1-Q3) with interquartile interval (IQR). Classification values are expressed in numbers (percentages). The categorical data were reported as frequency and percentage and compared using chi square or Fisher's exact test. The unpaired Student *t* test (if normal distribution) or Mann Whitney *U*-test (non-normal distribution variable) is used to compare the continuous variables between two independent groups. If more than two groups are compared, the ANOVA or Kruskal Wallis test is used for analysis. The logistic regression model was used for single-factor analysis to determine the correlation between different clinical factors and the occurrence of IMH. The baseline variables with statistical significance in the single-factor logistic regression analysis were included in the multivariate logistic regression analysis. Kaplan Meier curve was used to evaluate the difference in the incidence of endpoint events at 12 months (log rank method was used to calculate P value) post discharge. All tests were 2-tailed tests, and $P < 0.05$ was considered statistically significant. In this study, SPSS 26.0 (IBM Software Inc), EmpowerStats3.0 software and R (version 3.3.2) were used for statistical analysis, and R (version 3.3.2), Graph Pad Prism V8.0 (GraphPad Software Inc) and PowerPoint 2019 (Microsoft Inc) were used for mapping.

Results

Clinical Features of STEMI Patients According IMH

A total of 40 STEMI patients were enrolled, and 34 (85.0%) patients were male and 6 (15%) female, with a mean age of 55.9±10.3 years. Of these, 24 patients were in the IMH-absent group and 16 (40.0%) patients were in the IMH-present group. Patients in the IMH-present group had significantly higher serum levels of CRP (158.5±0.7 mg/L vs 30.6±3.8 mg/L, $P < 0.001$), HsTNT [2478.5 (1740.0–3805.2) pg/mL vs 296.0 (155.8–1894.5) pg/mL, $P = 0.009$] and CK-MB [150.8 (69.6–474.0) U/L vs 33.3 (19.2–68.7) U/L, $P = 0.011$] than those in the IMH-absent group. Regarding CMR findings, the IMH-present group had a significantly higher MI volume than the IMH-absent group (36.8±13.8 cm3 vs 20.7±14.7 cm3, $P = 0.003$), and LVEF was also significantly lower in the IMH-present group than the IMH-absent group (29.1±7.5% vs 40.4±11.6%, $P = 0.001$). In addition, the incidence of MVO was also significantly higher in the IMH-present group than in the IMH-absent group (75% vs 29.2%, $P = 0.004$). Other clinical characteristics such as demographics, treatment options, and laboratory findings are shown in Table 1.

Clinical Features of NSTEMI Patients According IMH

During the same period, 41 patients with NSTEMI were enrolled in the present study, and CMR was performed to determine whether IMH occurred before PCI. Thirty-one (75.6%) patients were male and with a mean age of 55.9±10.3 years. Three patients were in the IMH-absent group, and CMR imaging of the remaining patients did not reveal any suspicious IMH. Contrasting the differences in clinical characteristics between the two groups, serum CK-MB [165.9 (101.3–444.4) U/L vs 20.1 (15.9–27.7) U/L, $P < 0.001$], HsTNT [5893.0 (4230.0–7946.5) pg/mL vs 199.0 (44.0–658.0)

Table 1 Clinical Features of STEMI Patients According IMH

	All Patients (N = 40)	IMH Absent (n = 24)	IMH Present (n = 16)	*P*-Value
Age, years	55.9 (10.3)	56.4 (11.4)	55.2 (8.8)	0.727
Male, n (%)	34 (85)	21 (87.5)	13 (81.2)	0.588
Comorbidities, n (%)				
Smoking	30 (75.0)	17 (70.8)	13 (81.2)	0.456
T2DM	8(20.0)	4 (16.7)	4 (25.0)	0.690
Hypertension	17 (42.5)	9 (37.5)	8 (50.0)	0.433
Hyperlipidaemia	7 (17.5)	5 (20.8)	2 (12.5)	0.681
Medications, n (%)				
Aspirin	39 (97.5)	23 (95.8)	16 (100.0)	0.408
Clopidogrel	29(72.5)	20 (83.3)	9 (56.3)	0.109
LMWH	16(40.0)	11 (45.8)	5 (31.2)	0.356
Laboratory findings				
HGB, g/L	129.8(20.5)	127.8 (23.1)	133.0 (15.9)	0.434
WBC, 10^9/L	8.4(3.3)	8.4 (3.9)	8.3 (2.4)	0.946
PLT, 10^9/L	233.1(92.0)	236.5 (105.2)	227.9 (70.6)	0.776
TC, mmol/L	2.9(2.1)	2.7 (5.1)	3.2 (1.1)	0.435
TG, mmol/L	1.5(0.8)	1.5 (0.8)	1.6 (0.8)	0.835
LDL-C, mmol/L	2.4(0.8)	2.3 (0.7)	2.6 (0.8)	0.326
CRP, mg/L	44.1(51.4)	30.6 (3.8)	158.5 (0.7)	**<0.001**
CK-MB, u/L	52.3 (22.6–192.3)	33.3 (19.2–68.7)	150.8 (69.6–474.0)	**0.013**
HsTnT, pg/mL	1710.0 (197.8–2826.2)	296.0 (155.8–1894.5)	2478.5 (1740.0–3805.2)	**0.009**
NT-proBNP, pg/mL	2006.0 (999.8–4179.2)	2006.0 (1153.8–4411.5)	1727.5 (901.8–3585.2)	0.516
Killip-class on admission, n (%)				0.191
I	8 (20.0)	4 (16.7)	4 (25.0)	
II	11 (27.5)	5 (20.8)	6 (37.5)	
III	14 (35.0)	9 (37.5)	5 (31.2)	
IV	7 (17.5)	6 (25.0)	1 (6.2)	
Infarct related artery, n (%)				
Left anterior descending	19 (47.5)	9 (37.5)	10 (62.5)	0.121
Left circumflex	12 (30.0)	8 (33.3)	4 (25.0)	0.573
Right coronary artery	8 (20.0)	7 (29.2)	1 (6.2)	0.076
Left main	1 (2.5)	0 (0.0)	1 (6.2)	0.215
Spontaneously reperfused	23(57.5)	12 (50.0)	11 (68.8)	0.240
Echocardiographic findings				
LAESd, mm	38.7 (5.3)	40.0 (6.0)	37.0 (3.6)	0.087
RAESd, mm	33.4 (4.2)	59.5 (10.4)	56.6 (6.9)	0.346
LVEDd, mm	58.3 (9.2)	34.4 (4.8)	32.1 (2.8)	0.091
RVEDd, mm	32.7 (4.2)	33.1 (3.8)	32.1 (4.9)	0.472
LVEF, (%)	43.0 (12.4)	45.6 (13.3)	39.2 (10.2) 3	0.112
CMR findings, n (%)				
MVO	19 (47.5)	7 (29.2)	12 (75.0)	**0.004**
Ventricular aneurysm	17 (53.1)	8 (33.3)	9 (56.2)	0.094
Infarction volume, cm^3	26.3 (15.8)	20.7 (14.7)	34.6 (13.8)	**0.003**
IMH volume, cm^3	–	–	5.3 (3.8)	–
LVEF (CMR), (%)	35.8 (11.5)	40.4 (11.6)	29.1 (7.5)	**0.001**

Notes: Data are (N) Mean (SD) or (N) n (), Median (Q3-Q1), where N is the total number of patients with available data. Boldface indicates statistical significance ($P < 0.05$).

Abbreviations: T2DM, type 2 diabetes mellitus; LMWH, low molecular weight heparin; CMR, cardiac magnetic resonance; TC, total Cholesterol; TG, Triglyceride; LDL-C, low density lipoprotein-cholesterol; LAESd, Left Atrium End Systolic diameter; LVEDd, Left Ventricular End Diastolic diameter; RAESd, Right Atrium End Systolic diameter; RVEDd, Right Ventricular End Diastolic diameter; LVEF, Left Ventricular Ejection Fraction; IMH, intramyocardial hemorrhage; MVO, microvascular obstruction.

pg/mL, $P < 0.001$], and NT-proBNP [2983.0 (1962.5–18,991.5) pg/mL vs 1589.0 (840.0–2816.2) pg/mL, $P = 0.013$] were significantly higher in the IMH present group than in the IMH absent group. Clinical features as well as CMR findings of the overall patients, as well as according to IMH status, are presented in Table 2.

Table 2 Clinical Features of NSTEMI Patients According IMH

	All Patients (N = 41)	IMH Absent (n = 38)	IMH Present (n = 3)	*P*-Value
Age, years	62.6 (10.8)	62.2(11.1)	67.3(6.4)	0.437
Male, n (%)	31(75.6)	28 (73.7)	3 (100.0)	0.307
Comorbidities, n (%)				
Smoking	26(63.4)	23(60.5)	3(100.0)	0.456
T2DM	15 (36.6)	14(36.8)	1(33.3)	0.903
Hypertension	28 (68.3)	26(68.4)	2(66.7)	0.950
Hyperlipidaemia	8(19.5)	8(21.1)	0(0.0)	0.376
Medications, n (%)				
Aspirin	39 (95.1)	36 (94.7)	3 (100.0)	0.684
Clopidogrel	10 (24.4)	10 (26.3)	0 (0.0)	0.307
LMWH	22 (53.7)	20 (52.6)	2 (66.7)	0.639
Laboratory findings				
HGB, g/L	130.2(22.7)	130.2(23.0)	130.0(22.6)	0.986
WBC, 10^9/L	7.6(1.4)	7.5(1.3)	8.3(2.3)	0.740
PLT, 10^9/L	224.7(74.8)	225.9(76.8)	209.0(49.8)	0.712
TC, mmol/L	3.8(0.9)	3.8(0.9)	4.4(0.6)	0.158
TG, mmol/L	1.5(0.9)	1.5(0.9)	1.6(0.8)	0.728
LDL-C, mmol/L	2.3(0.8)	2.3(0.8)	2.5(0.5)	0.596
CRP, mg/L	40.0(68.6)	36.7(67.0)	147.0(74.0)	0.115
CK-MB, u/L	20.4 (16.3–42.1)	20.1(15.9–27.7)	165.9 (101.3–444.4)	**<0.001**
HsTnT, pg/mL	210.0 (46.0–1004.5)	199.0 (44.0–658.0)	5893.0 (4230.0–7946.5)	**<0.001**
NT-proBNP, pg/mL	1655.0 (909.5–3030.5)	1589.0 (840.0–2816.2)	2983.0 (1962.5–1991.5)	**0.013**
Grace scores				0.781
≤108	16 (39.0)	15 (39.5)	1 (33.3)	
109–140	21 (51.2)	19 (50.0)	2 (66.7)	
>140	4 (9.8)	4 (10.5)	0 (0.0)	
Three branches lesion	27 (65.9)	25 (65.8)	2 (66.7)	0.975
SYNTAX scores	40.5(9.3)	39.6(9.7)	46.0(4.0)	0.279
Killip-class on admission, n (%)				0.358
I	5 (12.2)	5 (13.2)	0 (0.0)	
II	16 (39.0)	15 (39.5)	1 (33.3)	
III	11 (26.8)	11 (28.9)	0 (0.0)	
IV	9 (22.0)	7 (18.4)	2 (66.7)	
Echocardiographic findings				
LAESd, mm	39.2(6.2)	39.4(6.2)	37.3(7.2)	0.592
RAESd, mm	32.2(6.9)	32.2(7.0)	33.0(7.0)	0.847
LVEDd, mm	56.8(8.2)	56.6(8.1)	59.0(11.1)	0.629
RVEDd, mm	31.3(3.7)	31.4(3.6)	30.3(5.0)	0.644
LVEF, (%)	44.3(12.6)	44.1(12.5)	46.3(17.8)	0.774
CMR findings, n (%)				
MVO	13 (31.7)	10 (26.3)	3 (100)	0.844
LVEF (CMR), (%)	39.1(15.2)	38.9(15.0)	40.4(20.8)	0.873

Notes: Data are (N) Mean (SD) or (N) n (), Median (Q3-Q1), where N is the total number of patients with available data.
Abbreviations: NSTEMI, non-ST-segment elevation myocardial infarction. Boldface indicates statistical significance (P < 0.05).

Outcome of STEMI Patients According IMH

These patients were followed up for a median time of 13 (11–13) months, and 7 STEMI patients [death, n = 1; new congestive heart failure, n = 4; reinfarction, n = 2]. And 12 NSTEMI patients [death, n = 2; new congestive heart failure, n = 8; reinfarction, n = 2] experienced a MACE event. Among STEMI patients, 5 (5/16) patients in the IMH present group and 2 (2/24) in the IMH absent group experienced MACE events. The log rank test was performed at the 12 months after discharge, and patients in the IMH present group had a worse prognosis than those in the IMH absent group (p = 0.063) (Figure 2). Regarding NSTEMI patients, 1 (1/3) patients in the IMH present group experienced MACE events, because of the small sample size, it was not possible to compare the prognostic differences between the two groups.

Incidence of IMH According to Different Types of Clinical Conditions

The incidence of IMH was significantly higher in patients with STEMI who did not receive reperfusion therapy than in patients with NSTEMI (16/40 vs 3/41, P < 0.001) (Figure 3a). To contrast the incidence of IMH in patients with STEMI who received reperfusion therapy, we also collected relevant medical information on patients with STEMI who were admitted to our hospital during the same time period and underwent primary PCI and CMR. Sixty-five patients underwent primary PCI and 27 patients presented with IMH. Specific clinical characteristics of these patients are shown in Supplementary Table. Among patients with STEMI, the incidence of IMH was not significantly different between patients who with or without underwent PPCI (16/40 vs 27/65, p = 0.876) (Figure 3b). To examine the correlation between spontaneous reperfusion and the occurrence of IMH in STEMI patients who did not receive reperfusion therapy. Patients in the spontaneous reperfusion group

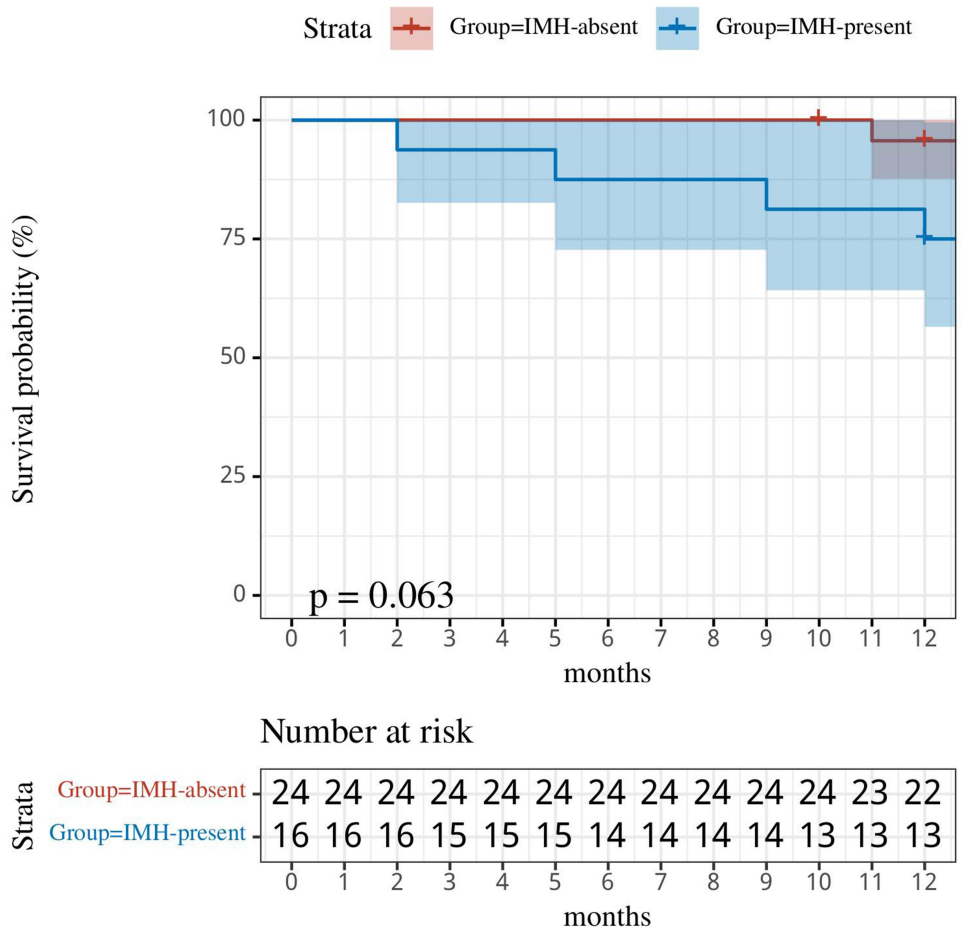

Figure 2 The Kaplan–Meier curve was used to compare the MACE-free survival in the IMH-present group and the IMH-absent group patients. The rate of MACE events at 12 months in IMH-present group was higher than that in IMH-absent group (5/16 vs 2/24, P = 0.063).

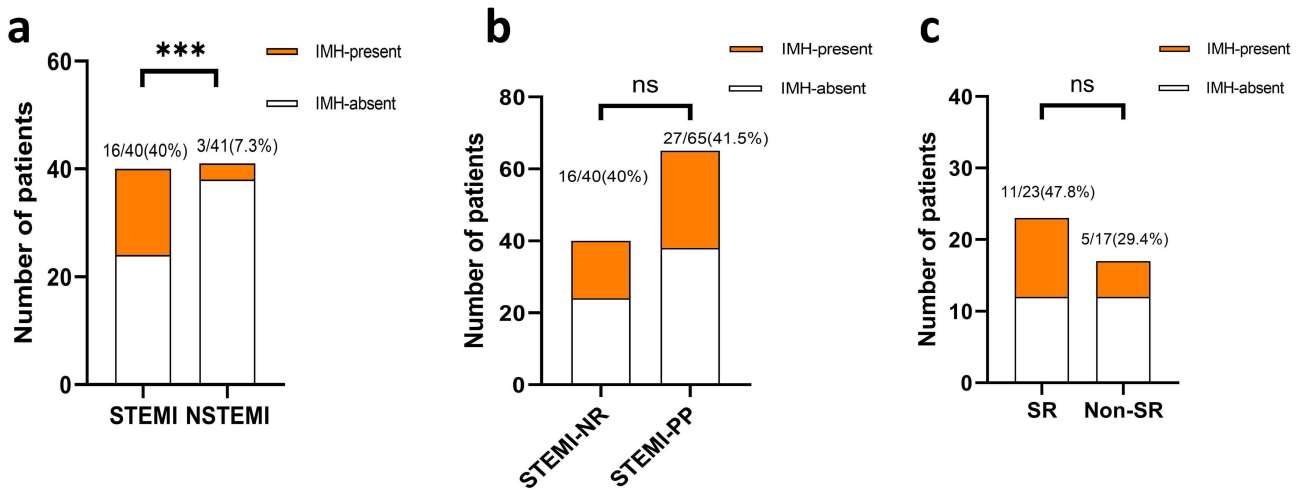

Figure 3 (**a**) The incidence of IMH was significantly higher in patients with STEMI who did not receive reperfusion therapy than in patients with NSTEMI (41.5% vs 7.3%, P < 0.001); (**b**) Among patients with STEMI, the incidence of IMH was not significantly different between patients who with or without underwent PPCI (16/40 vs 27/65, p = 0.876); (**c**) Patients in the spontaneous reperfusion (SR) group had a higher incidence of IMH than patients in the non-SR group (11/23 vs 5/17, P = 0.240).

had a higher incidence of IMH than patients in the non-spontaneous reperfusion group, but the difference did not reach statistical significance (11/23 vs 5/17, P = 0.240) (Figure 3c).

Discussion

Previous studies have suggested that IMH is a kind of myocardial abnormal pathological tissue after reperfusion therapy in patients with STEMI, which may be caused by ischemia-reperfusion injury to the myocardium and coronary vessels, and associated with poor outcomes.[6,12] Although reperfusion therapy is already the best treatment for AMI, there are still considerable patients in developing countries and remote areas who miss the timing of reperfusion because of patient delays or delays in first aid. Based on the results of the CAMI registry study,[13] the rate of receiving PPCI for STEMI patients in China was less than 80%, and the rate of reperfusion therapy (PCI) for NSTEMI patients was even lower.[14] Therefore, the findings regarding IMH in this subset of patients contribute to better understanding of the relationship between reperfusion therapy and IMH.

First of all, we first revealed that patients with NSTEMI can also occur IMH, but its incidence is far lower than that of patients with STEMI. The present study examined 41 patients with NSTEMI by CMR and only 3 patients presented with IMH, and due to the small sample size, the incidence of IMH in NSTEMI cannot be systematically described. In addition, we lack data on IMH in NSTEMI patients receiving PCI resulting in the inability to compare the difference in the incidence of IMH between the two groups. We found that these 3 patients with NSTEMI who developed IMH were mainly seen with myocardial infarction in the anterior wall of the left ventricle (LV) supplied with a large MI volume, and 2 had a previous history of coronary angina and 1 had a combined history of diabetes. So, we conjectured whether IMH occurred in patients with NSTEMI, mainly related to MI volume and the narrow culprit vessel. Left coronary ischemia leads to anterior LV infarction, and myocardial infarction volume is larger. This speculation requires a larger sample size to investigate the clinical characteristics of IMH in NSTEMI while continuing to analyze whether IMH in patients with NSTEMI has an adverse prognostic impact.

Secondly, the present study revealed that the incidence of IMH was similar in patients with STEMI with or without reperfusion therapy. IMH was previously thought to result after a slightly later reperfusion treatment, early reperfusion, therefore, can salvage the myocardium when the endothelium is still intact, whereas later reperfusion might result in erythrocyte extravasation to already necrotized myocardium and have reduced or no benefit.[15–17] However, our investigation suggests a similar incidence of IMH whether receiving reperfusion therapy or not, which is puzzling. Vargas et al[18] first reported that IMH not only occurred after reperfusion therapy but was also seen in patients with spontaneous reperfusion. Therefore, we analyzed coronary angiography (CAG) recordings during in-hospital elective PCI and searched for the precise culprit vessel by CMR of the LGE corresponding MI site, while we compared spontaneous reperfusion of infarct-related vessels with IMH (Table 1). Spontaneous reperfusion of the infarct-related artery was defined as the state in which the patient's

infarct-related arteriogram showed normal patency and reached TIMI grade 2–3. Subsequently, we further found that the incidence of IMH was higher in patients in the spontaneous reperfusion (SR) group than in those in the non-SR group, although the difference did not reach statistical significance, and we believe that when the sample size increases, a statistical difference will be reached. The study of *Vargas* et al was a small sample size study with a case number of only 7 patients, and all 7 patients developed spontaneous reperfusion, including two patients who developed IMH. We have further analyzed this condition and brought some different understandings. Even patients with STEMI without reperfusion treatment can still present with IMH, IMH can occur regardless of whether spontaneous reperfusion occurs, but spontaneous reperfusion is associated with a higher incidence of IMH. Although there is no blood flow to the core region of the MI, whether these IMH originate from residual blood flow from the original coronary vessel or result from flow from other collateral circulations requires further study to explain this phenomenon. This phenomenon suggests that there may be other mechanisms involved in the generation of IMH that remain to be understood and will require further investigation.

The present study has some limitations. First of all, the sample size was small, in particular, a larger sample size of patients with NSTEMI is missing to explore whether IMH has an impact on the prognosis of patients with NSTEMI. Secondly, in this study, CMR was performed only once in all patients, at a suitable time after the patient was admitted to the hospital, it does not fully reflect the occurrence of IMH in patients. Although serial assessment in patients with CMR would have been ideal, repeated imaging studies of patients before and after PCI separately will also be clinically challenging and potentially unethical.

Conclusions

The incidence of IMH was significantly higher in patients with STEMI who did not receive reperfusion therapy than in patients with NSTEMI. The incidence of IMH is comparable in patients with STEMI with or without reperfusion therapy. STEMI patients can present with IMH even when infarct-related vessel blood flow was not restored.

Data Sharing Statement

Data available on reasonable request from the authors.

Ethics Approval and Consent to Participate

The study protocol conformed to the ethical guidelines of the Declaration of Helsinki as reflected by prior approval from the human research and ethics committee of the Second Xiangya Hospital of Central South University, the ethics approval number is (Y2021453-2). In China, for patients with acute myocardial infarction, the right to informed consent is often authorized to family members, except in cases where patients have mild symptoms and are fully conscious. This is largely due to cultural practices that prioritize family involvement in critical medical decisions, especially when patients are in a compromised state and may be unable to make complex decisions independently. In these situations, families play a key role in supporting the patient's welfare and helping to make urgent medical choices.

Consent for Publication

All authors declare that the submitted work is original and has not been published before (either in English or any other language) and that the work is not under consideration for publication elsewhere.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors have no conflicts of interest to disclose in this work.

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