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Original Article

The prognosis of Different Types of Reciprocal ST-segment Depression (R-ST-D) on Electrocardiograms in Acute Myocardial Infarction

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SUMMARY

Background: We investigated how to define the culprit coronary artery according to different reciprocal ST-segment depression (R-ST-D) *types* on electrocardiograms in ST-segment elevation acute myocardial infarction (STEMI), as well as the high-risk factors.

Methods: To analyze the prognosis of different R-ST-D types to define the culprit coronary artery and high-risk factors, 967 patients with STEMI were included and divided into four groups according to STEMI infarction sites and R-ST-D type: group I (type I), without R-ST-D (n = 143); group II (type II), R-ST-D less than or equal to the amplitude of ST-segment elevation (n = 664); group III (type III), R-ST-D greater than or equal to the amplitude of ST-segment elevation (n = 93); and group IV (type IV), the amplitudes of R-ST-D and ST-segment elevation were both elevated (n = 67).

Results: The incidence of type II was the highest at 68.7%, followed by that of type I, which was mainly due to anterior descending branch stenosis. Type IV was mainly caused by complete occlusion of multiple vessels including the anterior descending branch and circumflex branch and/or right coronary artery. Type III was always related to a higher incidence of malignant complications, ventricular wall motion disorders, and ejection fraction index ≤50% compared with types I and II ($p < 0.05$ and $p < 0.01$, respectively).

Conclusion: Different high-risk stratifications of R-ST-D in patients with STEMI, especially type III and IV, can be used as objective independent indices to predict and assess the culprit coronary artery and life-threatening prognosis.

1. Introduction

Coronary heart diseases (CHDs) are among the main diseases with serious life-threatening complications and effects on the quality of life. ST-segment elevation acute myocardial infarction (STEMI) is the most serious type of CHD, and it is characterized by a high mortality rate, high complication rate, and life-threatening prognosis.[1,2] As there are different electrocardiogram (ECG) characteristics for the different stages of STEMI, different characteristic changes of reciprocal ST-segment depression (R-ST-D) can be used to identify post-STEMI cardiovascular events in a timely manner and to identify populations with a high risk of sudden death.[3,4] Studies that focus on the different types of R-ST-D in patients with STEMI and their relationships with the culprit coronary artery and high-risk stratifications of clinical prognosis are rare.[5] Therefore, we investigated the relationships of different types of R-ST-D with the culprit coronary artery and high-risk stratifications of clinical prognosis in order to provide objective evidence for clinical treatments.

2. Materials and methods

2.1. Study population

Nine hundred sixty-seven patients with STEMI and complete clinical data at our hospital, from January 2001 to December 2012, were included in this retrospective analysis. The patients were divided into four groups according to their degree of R-ST-D amplitude offset: type I, the STEMI R-ST-D amplitude showed no depression (143 patients, including 83 men and 60 women aged about 79 years [53.3 ± 6.7 years]); type II, the STEMI R-ST-D amplitude was less than or equal to the amplitude of STE-IR (664 patients, including

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395 men and 269 women aged 33–81 years [55.3 ± 6.3 years]); type III, the STEMI R-ST-D amplitude was more than the amplitude of STE-IR (93 patients, including 52 men and 41 women aged 31–83 years [57.3 ± 6.9 years]); and type IV, the amplitudes of STEMI R-ST-D and STE-IR were both elevated (67 patients, including 39 men and 28 women aged 37–77 years [54.3 ± 6.3 years]). As the differences in sex and age among the four groups were not significant, the groups were comparable. Patients with combined diseases such as a bundle branch block, ventricular pre-excitation syndrome, or ECG-detected diseases were excluded. Serum biochemical markers of myocardial necrosis (troponin l, troponin T, and creatine kinase-MB), echocardiography findings, and ECG findings were routinely assessed. All patients underwent percutaneous coronary angioplasty and stenting. This study was conducted in accordance with the declaration of Helsinki. This study was conducted with approval from the Ethics Committee of Yantai Hospital. Written informed consent was obtained from all participants.

2.2. ECG evaluation

The PageWriter TC 50 electrocardiography system (Philips Healthcare) was used, and the ECG time points were as follows: 1) immediately on admission to the hospital, 2) within 8 h of acute myocardial infarction (AMI) onset, 3) before stenting, 4) within 6 h after stenting, 5) 12 h after stenting, and 6) ≥24 h after stenting. The PR-segment was set as the measurement baseline, and the ST-segment was measured at the J point. A limb lead elevation of ≥0.1 mV and chest lead elevation of ≥0.2 mV were defined as ST-segment elevation (STE). A Q wave duration of ≥40 ms and depth of ≥0.1 mV were defined as pathological Q wave. The pathological types were determined based on a comparison of the R-ST-D amplitude with the corresponding STE amplitude. The corresponding parts were as follows: the lead in the front wall or high lateral wall (I, aVL, and v1–v5) corresponded to that in the lower wall (II, III, and avF), and the rear wall (v7, v8, and v9) corresponded to the anterior partition (v1, v2, and v3). ST-segment depression (ST-D) ≥0.5 mV was considered abnormal, and at least two or more of the aforementioned changes were considered to occur in a consecutive lead. An ECG was used to categorize the infarction site, ΣST elevation amplitude, leads with STE, post-stenting R-ST-D changes, clinically concurrent pump failure, hypotension, severe arrhythmia, AMI extension, ventricular wall motion disorders, low left ventricular ejection fraction, and mortality.

2.3. Diagnostic criteria

All patients met the World Health Organization diagnostic criteria for CHD,[6,7] and they exhibited AMI-STE and dynamic increases in the serum biochemical markers of myocardial necrosis. According to the status of coronary artery stenosis, coronary lesion vessels and the culprit coronary artery were categorized into at least one major coronary artery, with ≥75% stenosis in the major branches or 100% occlusion (i.e., single-vessel lesion, double-vessel lesion, and triple-vessel lesion). The lesion was determined according to the left anterior descending branch (LAD), left circumflex branch (LCX), and right coronary artery (RCA). AMI of the front wall included the anterior partition, anterior lateral partition, and high lateral wall, whereas AMI of the lower wall included the rear wall and right ventricle.

2.4. Statistical analysis

The SPSS 12.0 statistical package (SPSS Inc., Chicago, IL, USA) was used to perform all statistical analyses. The measurement data are expressed as a mean ± standard deviation. Intergroup comparisons were analyzed using the *t*-test. The count data were assessed using the χ^2 test. A p value of <0.05 was considered statistically significant.

3. Results

3.1. Comparison of the incidence, culprit coronary artery stenosis, and coronary lesions

The results showed that the incidence of type II was the highest among all the types. Types III and IV mainly exhibited LAD- and RCA-based multivessel lesions, and the incidence of complete coronary artery occlusion in these groups was the highest ($p < 0.05$ and $p < 0.01$, respectively). Type I mainly exhibited LAD-based single-vessel lesions (Table 1).

3.2. Comparison of AMI sites, ΣSTE amplitude, leads with STE, and R-ST-D changes

Type II exhibited the highest incidence of R-ST-D changes ≤6 h after stenting ($p < 0.05$ to $p < 0.01$; Table 2), followed by type III.

3.3. Comparison of clinical complications

Patients with types III and IV had clinically diagnosed pump failure, hypotension, severe arrhythmia, AMI extension, ventricular wall motion disorders, and a low ejection fraction index; additionally, the mortality rate was higher for these types than for type I ($p < 0.05$ and $p < 0.01$, respectively; Table 3).

4. Discussion

Some patients with early STEMI may exhibit myocardial infarction corresponding with R-ST-D on their ECG. It was previously believed that R-ST-D represents STE-IR[8] and that it has no clinical significance. Recently, studies on radionuclide ventriculography, percutaneous transluminal coronary angioplasty (PTCA), echocardiography, enzymology, and autopsy found that this kind of ST change is caused by the corresponding myocardial ischemia and/or inferior endocardial infarction, and it is directly associated with an infarcted coronary occlusion.[9–11] This study shows that the ECG of patients with early STEMI exhibited four types of changes in R-ST-D corresponding to the infarcted anatomic sites, with varying high-risk prognoses among patients. The incidence of type I was 14.8%, and only some patients exhibited complete LAD occlusion-based single-vessel disease; the incidence rates of AMI in the front and complex front wall and the lower wall were 72.7% and 27.3%, respectively. The clinical complications of these patients were relatively fewer than that of patients in the other groups, and their short-term prognosis was good. The mechanism of R-ST-D does not change, and either no stenosis or less stenosis (<75%) in the corresponding infarcted coronary zone occurs. Although severe multivessel stenosis (>80%) existed and the corresponding infarcted coronary zone had good collateral blood supply, patients had a corresponding infarcted zone in the lower wall. Therefore, if the RCA exhibits severe stenosis, the circumflex artery will be coarse and vice versa. When the front wall is the corresponding zone of infarction and if the middle or distal LAD exhibits severe stenosis, the diagonal branch and obtuse branch will be thick and enriched with collateral branches to reach the distal blood supply of the diseased vessels; this is why blood vessels in the corresponding region of infarction have stenosis and R-ST-D does not occur. The occurrence of R-ST-D is highly associated with multiple stenoses in the front wall. The incidence rate of type II was 68.7%, and the incidence rates of AMI in the front or/and complex front wall and

Table 1
Comparison of incidence, culprit coronary artery stenosis, and coronary lesions among different R-ST-D type groups (n, %).

Group		Incidence	Stenosis	Coronary lesions			Culprit coronary artery		
	(%)	Complete occlusion	Incomplete occlusion	1	2	3	LAD-based	LCX-based	RCA-based
I (n = 143)	14.8	109 (76.2)	34 (23.8)	113 (79.0)	24 (16.8)	6 (4.2)	97 (67.8)	7 (4.9)	39 (27.3)
II (n = 664)	68.7[b]	435 (65.5)	229 (34.5)	316 (47.6)	216 (32.5)[b]	132 (19.9)[b]	397 (59.8)	98 (14.8)[b]	169 (25.4)
III (n = 93)	9.6	87 (93.5)[a]	6 (6.5)	0	24 (25.8)	69 (74.2)[bd]	51 (52.7)	10 (10.7)[a]	32 (34.4)
IV (n = 67)	6.9	65 (97.0)[a]	2 (3.0)	0	19 (28.4)[a]	48 (71.6)[bd]	34 (50.8)	9 (13.4)[b]	24 (35.8)

Note: compared with group I: [a]P<0.05, [b]P < 0.01; compared with group II: [c]P < 0.05, [d]P < 0.01; compared with group III: [e]P < 0.05, [f]P < 0.01.

Table 2
Comparison of AMI sites, ΣSTE amplitude, leads with STE, and R-ST-D evolution at different post-stenting time points among different groups (n, %).

Group (n)	AMI site			ΣSTE amplitude	STE Post-stenting	R-ST-D ≤6 h Evolution		
	Front wall	Lower wall	Front wall + lower wall	(mm)	Leads with STE	≤6 h	12 h	≥24 h
I (n = 143)	97 (67.8)	39 (27.3)	7 (4.9)	10.3 ± 1.1	4.1 ± 0.9	0	0	0
II (n = 664)	393 (59.2)	271 (40.8)[b]	27 (4.3)	9.6 ± 1.3	4.6 ± 1.1	519 (78.2)	129 (19.4)	16 (2.4)
III (n = 93)	21 (22.6)	17 (18.3)	55 (59.1)[bd]	11.8 ± 1.7[ad]	4.9 ± 1.9	0	14 (23.3)	77 (82.8)[d]
IV (n = 67)	0	0	67 (100)bdf	13.5 ± 2.2bdf	6.2 ± 2.3bdf	0	0	0

Note: compared with group I: [a]P<0.05, [b]P < 0.01; compared with group II: [c]P < 0.05, [d]P < 0.01; compared with group III: [e]P < 0.05, [f]P < 0.01.

Table 3
Comparison of clinical complications among the groups (n, %).

Group (n)	Pump failure	Hypotension	Severe arrhythmia	AMI extension	Mortality ventricular wall	Motion disorders	EF (≤50%)
I (n = 143)	11 (7.7)	13 (9.1)	26 (18.2)	3 (2.1)	5 (3.5)	61 (42.7)	13 (9.1)
II (n = 664)	41 (6.2)	64 (9.6)	97 (14.6)	93 (14.5)[a]	24 (3.6)	232 (34.9)	71 (10.7)
III (n = 93)	65 (69.9)[bd]	37 (39.8)[bd]	53 (57.0)[bd]	33 (35.5)[bd]	14 (15.1)[bd]	93 (100.0)[bd]	32 (34.4)[bd]
IV (n = 67)	48 (71.6)[bd]	28 (41.8)[bd]	41 (61.2)[bd]	23 (34.3)[bd]	11 (16.4)[bd]	67 (100.0)[bd]	27 (40.3)[bd]

Note: compared with group I: [a]P<0.05, [b]P < 0.01; compared with group II: [c]P < 0.05, [d]P < 0.01; compared with group III: [e]P < 0.05, [f]P < 0.01.

the lower wall were 59.2% and 40.8%, respectively. Furthermore, LAD- and RCA-based complete occlusions of single-vessel or double-vessel disease were common. Additionally, clinical complications occurred relatively less frequently, and the short-term prognosis was good. Changes within 12 h after stenting were the highest ($p < 0.05$ or $p < 0.01$), and with STE in the infarcted zone, this gradually decreased. The R-ST-D also gradually returned to baseline, showing a clear positive correlation between R-ST-D and stenting. The following mechanisms have been considered. 1) The mirror reaction: during PTCA surgery, it was found that when the balloon occludes one single vessel, the R-ST on ECG shows an increase in this vessel-dominated region; additionally, many patients exhibit the corresponding R-ST-D,[12,13] which is similar to that found in our patients. However, transient ST-D is positively associated with the degree of STE of the infarcted region and minor changes. 2) Myocardial ischemia: in anterior wall AMI, when the ST-segment of the inferior wall is depressed, the incidence of RCA occlusion combined with ST-D is 80%; thus, the corresponding lead indicates ischemic ST-segment changes, and is related to the gradual return to normal of the R-ST-D due to the recovery of collateral circulation or blood supply to the corresponding region within 12 h of revascularization of the infarcted coronary artery. Recent studies have shown that R-ST-D in the distal infarcted zone is mostly caused by an expanded infarction area; thus, the distinguishing mirror changes and infarct expansion depend on the time and amplitude of ST-D. If ST-D appears within a few hours of AMI and lasts for >24 h with an amplitude >0.45 mV, it is often caused by infarct expansion; otherwise, it is caused by mirror changes.[14,15] The incidence rate of type III was 9.6%; the incidence rates of AMI in the front or/and complex front wall and the lower wall were 81.7% and 18.3%, respectively. The degree and duration of ultra-low R-ST-D, and the severity of myocardial ischemia are associated with a high-risk prognosis. A higher STE amplitude in the infarcted region, higher number of leads, and longer R-ST-D duration (≥24 h) cause a larger infarct size. On the other hand, the existence of severe myocardial ischemia distal from the infarcted site often causes multivessel (LAD and RCA) or triple-vessel coronary artery disease. The higher the risk of infarction expansion, the more obvious the ST-D. Additionally, the more diseased the coronary arteries, the more severe the stenosis. The complications are mainly related to the severity of the myocardial injury, and the important and reliable indicator of prognosis is the ejection fraction index. Myocardial injury is associated with specific complications. Recent studies have shown that the in-hospital mortality rate of patients with STEMI younger or older than 75 years decreased from 11.5% to 8.0%, and this may be associated with the diagnosis, treatment measures, and related symptoms of sudden cardiac events.[16] When recovery at 12 h post-stenting is poor, the R-ST-D will not gradually recover with STE in the infarcted region; additionally, if the recovery baseline exhibits one negative correlation, the ultra-depression and duration of ST will poorly match the STE. Thus, large infarcts and critical conditions are important prognostic indicators for diagnosing high-risk subgroups of patients with STEMI, the prognosis of whom will be severe.[17] Acute infarcted coronary lesions and severe coronary stenosis until total occlusion in the corresponding parts mainly show minor non-STEMI (NSTEMI) changes during chronic degenerative processes and are due to additive effects.[18] The incidence of type IV was 6.9%, and this type had the highest incidence of multivessel and complex front wall diseases such as LAD or LCX-RCA total occlusion. The higher the STE amplitude, the higher the number of leads,[5,17] the more severe the degree of multivessel myocardial injury, the greater the range of complications, the worse the cardiac function, and the higher the number of patients who will be prone to a poor prognosis of cardiac emergencies. The main complications of STEMI are related to the extent and scope of myocardial injuries in the infarcted region and corresponding

region. Therefore, the more significantly the ejection fraction index decreases, the higher will be the incidence of AMI-related complications and emergent events, including pump failure, hypotension, severe arrhythmia, AMI extension, and high mortality. This may be related to multipart AMI, especially the combination of complete left bundle branch block,[1,19] which leads to elevation of the infarcted zone and R-ST-D (the independent risk factor of a short-term malignant prognosis). Recent studies have shown that more than one-fourth of Chinese patients with AMI have more than three correctable cardiovascular risk factors; more than one-half of patients with AMI have a history of smoking, overweight/obesity, and hypertension; and nearly 80% of patients with AMI have an unhealthy lifestyle such as a fatty diet and lack of exercise.[20] In brief, the mechanisms of R-ST-D in patients with STEMI are complicated, as they can present as mirror changes, an infarcted area, and extensive changes, or NSTEMI can occur distal from the site. We believe that the classification of R-ST-D can help physicians objectively assess patients with a high-risk prognosis, and that types III and IV are independent risk factors of short-term cardiovascular outcomes in patients with STEMI. Moreover, we believe that regular high-risk stratification can predict the short-term clinical prognosis.

Competing interests

The authors declare that they have no competing interests.

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