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Coronary Artery Disease

The Difference on Features of Fragmented QRS Complex and Influences on Mortality in Patients with Acute Coronary Syndrome

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Objectives: To investigate whether the fragmented QRS (fQRS) complexes can be used to distinguish patients with early non-ST elevation myocardial infarction (NSTEMI) from those with unstable angina (UA).
Background: fQRS complex has been found to be linked to myocardial infarction and cardiac death.
Methods: The clinical data of 302 patients who had been diagnosed with coronary artery disease were retrospectively reviewed. Incidence of fQRS complex within 48 h of presentation was analyzed and patients with acute myocardial infarction (AMI) (n = 240) were followed up by telephone interviews for a mean of 61.47 (range, 59.60-63.35) months.
Results: Patients with NSTEMI exhibited higher incidence of fQRS than those with UA (p = 0.047). The incidence of fQRS in the inferior wall leads was significantly higher than that of other leads in patients with anterior wall infarction (p < 0.05). Kaplan-Meier analysis revealed a higher mortality rate in AMI patients with fQRS compared to non-fQRS patients (p = 0.001).
Conclusions: Presence of fQRS complexes within 48 hours of presentation may be used to differentiate NSTEMI patients from UA patients. fQRS may also be used as a survival predictor for patients with AMI.

Key Words: Acute coronary syndrome • Electrocardiography • Fragmented QRS • Non-ST elevation myocardial infarction • Unstable angina

INTRODUCTION

Acute coronary syndrome (ACS) is caused by myocardial necrosis or acute myocardial ischemia. It is usually diagnosed based on a full clinical assessment including a medical history review, the patient's clinical manifestations, echocardiographic and electrocardiographic findings, and/or the levels of cardiac enzymes.[1] ACS is categorized as unstable angina (UA, with no increase in cardiac enzymes), non-ST elevation myocardial infarction [NSTEMI; an increase in cardiac enzymes without ST elevation on 12-lead electrocardiography (ECG)], and ST elevation myocardial infarction (STEMI; ST elevation on ECG). STEMI is a consequence of the complete and lengthy occlusion of an epicardial coronary blood vessel, and is determined based on ECG criteria. NSTEMI usually occurs after the development of coronary artery narrowing, transient and partial occlusion, or microembolization of a thrombus and/or atheromatous material, and is determined by increased levels of circulating cardiac enzymes in the absence of ST elevation, whereas UA does not involve any significant elevation incirculating cardiac biomarkers. However, testing for cardiac enzymes may not be possible in resource-limited areas. In addition, clinicians tend to rely on a significant rise in cardiac enzyme levels to make a confident diagnosis, and it can be difficult to differentiate NSTEMI from

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UA if cardiac enzyme results are not readily available.

It has been shown that regional myocardial scars are associated with changes in QRS complex configuration that can cause terminal conduction delay or fragmented QRS (fQRS) complexes on 12-lead ECG,[2-4] the latter of which is defined as the presence of an additional R wave (R') or notching in the nadir of the S wave, or the presence of more than one R' in two contiguous leads, corresponding to a major coronary artery territory on resting 12-lead ECG (Figure 1). Alternatively, fQRS is also defined as the existence of different RSR' patterns with or without Q waves on resting 12-lead ECG.[5,6] Slow and non-homogenous conduction through ischemic or infarcted myocardium and conduction system has been postulated to be the mechanism of fQRS.[7,8] The presence of fQRS has been used to predict myocardial scars,[2,9] poor outcomes[7] and ventricular arrhythmias.[2,10]

Fragmented QRS complexes can be used as a marker of myocardial infarction and to predict cardiac events and cardiac death as a consequence of myocardial infarction.[11] In addition, the appearance of the fQRS complex is more sensitive than ST-T changes and pathological Q waves in the diagnosis of NSTEMI,[12] and fQRS has also been associated with previous silent myocardial infarctions, which are commonly seen in females with atypical chest pain, patients with diabetes mellitus, and in older patients with dementia.[2] In addition, a previous report suggested that susceptible patients with fQRS are at an increased risk of adverse cardiac events including acute myocardial infarction (AMI), revascularization, cardiac-related death, and even all-cause mortality.[7] Furthermore, it has been reported that patients with a history of Q wave MI have an increased risk of recurrent cardiac events including myocardial infarction if they also have fQRS.[11]

The early use of proper medications including antiplatelets and anticoagulants is critical for a favorable prognosis of NSTEMI.[13] However, it is difficult to differentiate patients with NSTEMI from those with UA if cardiac enzyme tests are not readily available. As the fQRS complex can be detected as early as several hours after AMI,[12] the present study aimed to explore the potential diagnostic value of the fQRS complex to diagnose NSTEMI within 48 hours of the onset of AMI.

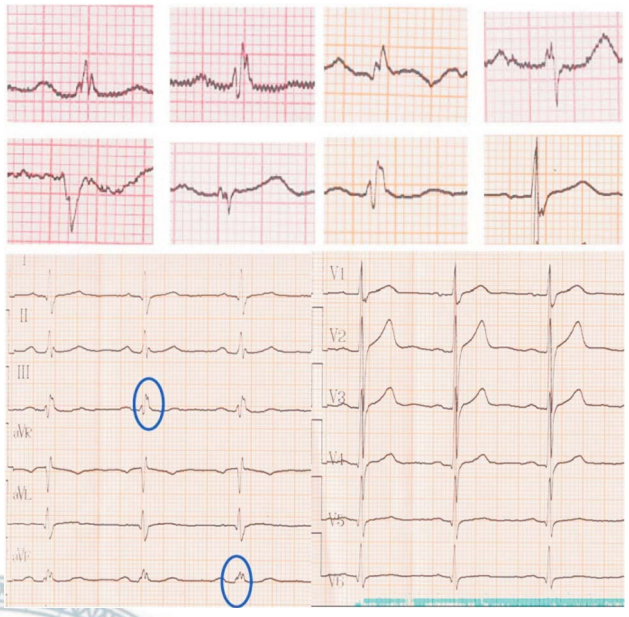

Figure 1. *Typical fQRS complex morphologies. The fQRS complexes are circled in blue.*

MATERIAL AND METHODS

Patients

Four hundred and seven patients with a diagnosis of coronary artery disease (CAD) treated at The Third People's Hospital of Hubei Province from January 2009 to December 2015 were reviewed. Among these patients, 227 (55.8%) were retrospectively reviewed. The diagnosis of ACS was made using associated risk factors, clinical symptoms, ECG changes, increases in cardiac biomarkers, and coronary angiography findings. NSTEMI and UA were differentiated using the cardiac marker, cardiac troponin I (ACS 180: SE System, Bayer, USA; reference interval: cTnI, 0~1.5 μg/L). Patients were excluded if they had any of the following conditions: a prior history of myocardial infarction (by taking their history and reviewing clinical records and previous ECG), complete or incomplete bundle branch block, pre-excitation syndrome and pacemaker implantation. The patients were diagnosed as having AMI, UA, and stable angina. AMI included NSTEMI and STEMI. This was a retrospective study and was approved by the Ethics Committee of The Third People's Hospital of Hubei Province, China.

ECG

ECG was performed in all patients at admission

within 48 hours of symptom presentation. All electrocardiograms were analyzed by the same cardiologist. Previous electrocardiograms were retrieved from the clinical records database and compared with the present findings to identify fQRS. The fQRS complex was defined by Das et al.[2] as the existence of an additional R wave (R') or notching in the nadir of the S wave, or the existence of more than one R' in two contiguous leads, corresponding to a major coronary artery territory on resting 12-lead ECG, with a QRS complex of less than 120 ms. The anterior wall was located using V1-V4 leads, and the lateral wall was located using I, avL, V5, and V6 leads. The inferior wall was located using II, III, and avF leads, and the right atrium was located using AVR, V3R, V4R, and V5R leads. The posterior wall was located using V7, V8, and V9 leads.

Coronary angiography

Coronary heart disease was diagnosed as stenosis ving detected by coronary angiography. Left anterior descending (LAD) lesions corresponded to the anterior and posterior ECG leads, right coronary artery (RCA) lesions corresponded to the inferior wall of the ECG leads and right ventricular leads, and left circumflex (LCX) lesions corresponded to the lateral leads.

Statistical analysis

Categorical data were reported as percentages, and continuous data as mean ± standard deviation (SD). Comparisons of continuous data were performed using one-way ANOVA, and comparisons of categorical data were performed using Fisher's exact test. SPSS version 18.0 (SPSS, Chicago, US) was used to perform the statistical analysis. Kaplan-Meier analysis was used to compare survival among the patients with and without fQRS. Risk factors for all-cause mortality were analyzed using Cox regression. Differences were considered to be statistically significant if the p value was less than 0.05.

RESULTS

Comparisons among the patients with different diagnoses

The patients identified and included in this study were recruited consecutively. Initially, 596 patients were identified, and 54 patients were excluded due to a history of previous myocardial infarction, 35 with bundle branch block, 2 with pacemaker rhythm, 105 with stable angina, and 98 who had incomplete clinical data. Thus, 302 patients were finally enrolled in this study (Table 1). The incidence rates of fQRS within 48 hours of presentation in the patients with STEMI or NSTEMI were higher than those with stable angina (p = 0.003 and p = 0.005, respectively). The patients with NSTEMI had a higher incidence of fQRS than those with UA (p = 0.047). In addition, there was no significant difference in the incidence of fQRS between the patients with and without early

Table 1. Comparison among patients with different diagnosis (n = 302)

	UA (n = 62)	STEMI (n = 187)	NSTEMI (n = 53)
Age (year)	61.2 ± 9.8	58.1 ± 13.1	60.9 ± 14.01
Male (n, %)	44 (71.0%)*	161 (86.1%)	44 (83.0%)
Hypertension (n, %)	34 (54.8%)	93 (49.7%)	33 (62.3%)
Diabetes (n, %)	10 (16.1%)	32 (17.1%)	13 (24.5%)
Smoker (n, %)	18 (29.0%)*#	115 (61.5%)	26 (49.1%)
Systolic pressure (mmHg)	131.7 ± 18.3*	120.5 ± 22.0#	132.5 ± 24.2
Diastolic pressure (mmHg)	79.3 ± 10.7	76.9 ± 13.3	79.6 ± 14.2
Total cholesterol (mmol/L)	4.1 ± 1.0	4.5 ± 1.2	4.5 ± 1.1
LVEDD (mm)	42.4 ± 9.6*#	45.9 ± 4.7	46.6 ± 4.1
LVEF (%)	55.1 ± 12.3*#	49.4 ± 7.9	50.7 ± 8.9
fQRS (n, %)	12 (19.4%)*#	67 (35.8%)	20 (37.7%)
QRS complex width (ms)	97.2 ± 5.6	95.4 ± 13.2	98.3 ± 13.3

fQRS, fragmented QRS; LVEDD, left ventricular end diastolic diameter; LVEF, left ventricular ejection fraction; NSTEMI, non-ST elevation myocardial infarction; STEMI, ST elevation myocardial infarction; UA, unstable angina.
* vs. STEMI; # vs. NSTEMI. All p < 0.05.

reperfusion treatment [8 (26.7%) vs. 79 (37.6%), p > 0.05]. Early reperfusion was performed using thrombolysis in 30 patients at 5.06 ± 1.60 hours. The sensitivity, specificity, positive and negative predictive values of fQRS in diagnosing NSTEMI were 37.7%, 64.5%, 62.5%, and 54.8%, respectively.

Comparisons between the patients with AMI with and without fQRS

Apart from a significant difference in the width of QRS complexes, there were no significant differences in clinical features and status of CAD between the patients with AMI with and without the fQRS complex within 48 hours of presentation (Table 2). These results suggest that fQRS was not caused by other demographic or clinical confounders.

Table 2. Comparison between AMI patients with and without fQRS complex within 48 h of presentation

	FQRS patients (n = 87)	Non-fQRS patients (n = 153)	p-value
Age (year)	61.4 ± 14.8	57.2 ± 12.2	0.026
Male (n, %)	74 (85.1%)	131 (85.6%)	0.905
Hypertension (n, %)	50 (57.5%)	76 (49.7%)	0.245
Diabetes (n, %)	19 (21.8%)	26 (17.0%)	0.355
Smoker (n, %)	51 (58.6%)	90 (58.8%)	0.976
Systolic pressure (mmHg)	120.8 ± 22.1	124.4 ± 23.5	0.241
Diastolic pressure (mmHg)	75.4 ± 13.0	78.7 ± 13.7	0.063
Total cholesterol (mmol/L)	4.5 ± 1.1	4.5 ± 1.1	0.873
LVEDD (mm)	46.2 ± 5.2	46.0 ± 4.1	0.835
LVEF (%)	47.8 ± 9.9	51.0 ± 6.4	0.044
QRS complex width (ms)	101.3 ± 15.0	93.1 ± 11.2	< 0.001

fQRS, fragmented QRS; LVEDD, left ventricular end diastolic diameter; LVEF, left ventricular ejection fraction.

fQRS in various leads in the patients with AMI

The incidence rates of fQRS complexes in various leads in the patients with AMI with single or multiple diseased vessels are shown in Table 3 and 4. The inferior wall leads showed a significantly higher incidence of fQRS (p < 0.05). No significant difference was observed in the incidence of fQRS among the leads of the anterior wall, lateral wall, and right atrium.

fQRS in the patients with various infarction locations

In the patients with anterior wall infarction, the inferior wall leads exhibited a higher incidence of fQRS [36 (25.5%)] compared to other leads (p < 0.05). The patients with inferior wall infarction had increased incidence rates of fQRS in the inferior wall, anterior wall, and right atrium leads. The patients with inferior wall infarction had a higher incidence of the fQRS complex in the right atrium leads than those with anterior wall infarction [14 (12.8%) vs. 5 (3.5%), p = 0.006] (Table 5).

Follow-up of the patients with AMI

The patients with AMI (n = 240) were followed up by telephone for a mean of 61.47 (range, 59.60-63.35) months. None of the patients were lost to follow-up. During the follow-up period, 12 patients died, of whom 9 had fQRS. Kaplan-Meier analysis showed that the patients with fQRS had a higher mortality rate than those

Table 3. The incidences of fQRS complexes in various leads in AMI patients with multiple diseased vessels

	Anterior wall leads	Inferior wall leads	Right atrium leads	Lateral wall leads	Any leads	p-value
LAD (n = 205)	15 (7.3%)	49 (23.9%)	13 (6.3%)	9 (4.4%)	67 (32.7%)	< 0.0001
LCX (n = 110)	9 (8.2%)	25 (22.7%)	6 (5.5%)	6 (5.5%)	38 (34.5%)	< 0.0001
RCA (n = 141)	11 (7.8%)	27 (19.1%)	9 (6.3%)	6 (4.3%)	42 (29.8%)	< 0.0001

AMI, acute myocardial infarction; fQRS, fragmented QRS; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery.

Table 4. The incidences of fQRS complexes in various leads in AMI patients with single diseased vessels

	Anterior wall leads	Inferior wall leads	Right atrium leads	Lateral wall leads	Any leads	p-value
LAD (n = 54)	3 (5.6%)	14 (25.9%)	3 (5.6%)	4 (7.4%)	17 (31.5%)	0.001
LCX (n = 11)	1 (9.1%)	1 (9.1%)	1 (9.1%)	2 (18.2%)	3 (27.3%)	0.879
RCA (n = 18)	1 (5.6%)	3 (16.7%)	1 (5.6%)	2 (11.1)	6 (33.3%)	0.628

AMI, acute myocardial infarction; fQRS, fragmented QRS; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery.

Table 5. Incidences of fQRS complex of various leads in patients with anterior or inferior wall infarctions

Infarction location	Anterior wall leads	Inferior wall leads	Right atrium leads	Posterior wall leads	Lateral wall leads	Any leads
Anterior wall (n = 141)	8 (5.7%)	36 (25.5%)	5 (3.5%)	1 (0.7%)	8 (5.7%)	48 (34%)
Inferior wall (n = 109)	12 (11.0%)	19 (17.4%)	14 (12.8%)	4 (3.7%)	3 (2.8%)	35 (32.1%)

without fQRS (p = 0.001) (Figure 2). Cox regression analysis identified the fQRS complex as an independent risk factor for all-cause mortality (p = 0.040) (Table 6).

DISCUSSION

In this study, the incidence of the fQRS complex within 48 hours of presentation in patients with AMI was significantly higher than that in patients with stable angina or UA, suggesting that the fQRS complex may be an additional risk factor for myocardial infarction. In addition, the patients with NSTEMI had a higher incidence of fQRS than those with UA. Furthermore, the patients with NSTEMI had a higher mean number of fQRS derivations compared to those with UA. Therefore, we believe that fQRS can be used to differentiate NSTEMI from UA among patients with ACS, at least within 48 hours of presentation. Our findings may prompt an increase in the use of early interventions for patients with NSTEMI, especially in resource-limited areas where cardiac enzyme tests are not readily available. We also found that patients with fQRS had a significantly higher rate of all-cause mortality, suggesting that fQRS can be used as a predictor of adverse outcomes in patients with CAD.

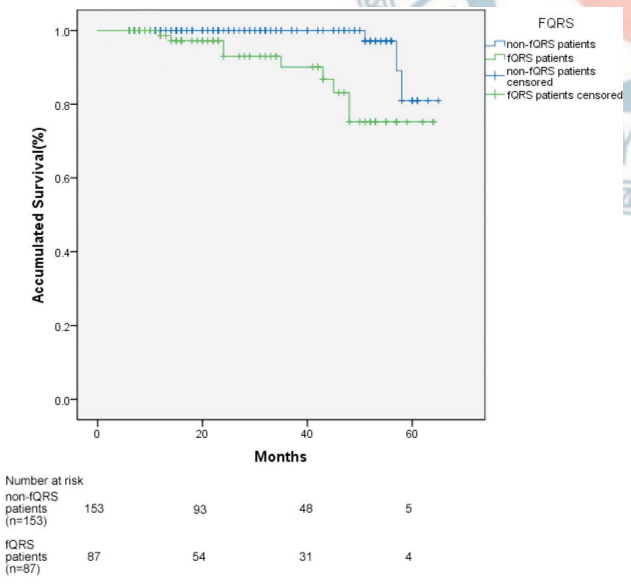

Figure 2. *Kaplan-Meier analysis of patients with AMI with and without fQRS. The patients with fQRS had a higher mortality rate than those without fQRS (p = 0.001). The number of survivors at each time point is shown below the figure.*

A more recent study showed that fQRS complexes were an independent predictor of major adverse cardiac events in patients with NSTEMI within 24 hours of presentation.[14] In the present study, we analyzed fQRS complexes within 48 hours of presentation, and found that the pattern of appearance of the fQRS complexes in CAD was significantly associated with the effective detection of fQRS. In order to accurately define the time course of the appearance of fQRS in patients with CAD, Das et al. examined the ECGs of 896 patients with ACS who underwent cardiac evaluation, and detected fQRS in 51% of the patients with myocardial infarction within 48 hours of presentation compared to only 3.7% of those with UA.[9] In addition, they also found that approximately half of the patients with NSTEMI developed fQRS within 48 hours of presentation.[12]

Non-homogeneous activation of the ventricles caused by myocardial scars or ischemia has been postulated to be one of the mechanisms underlying the development of the fQRS complex on resting 12-lead ECG.[15] The fQRS complex can also be defined as notching in the QRS wave after a myocardial infarction as a consequence of peri-infarction conduction block.[16] The potential mecha-

Table 6. Cox regression analysis of risk factors for all-cause mortality in AMI patients (n = 240)

	p-value	Risk ratio (95% CI)
Age	0.012	1.141 (1.029, 1.265)
fQRS	0.040	7.016 (1.097, 44.867)
Hypertension	0.522	NA
Diabetes	0.286	NA
Smoking	0.057	NA
Left ventricular ejection fraction	0.033	0.878 (0.779, 0.990)
Killip class ≥ 3	0.010	79.458 (2.830, 2231.149)

nism of fragmentation has been reported in autopsy findings in patients with myocardial infarction and left ventricular aneurysm, in whom severe myocardial necrosis with clusters of viable myocardial tissue intermixed in the fibrous tissue was observed.[4,17-19] Myocardial regions of chronic ischemia have slow activation due to partial depolarization and decreased velocity of the action potential upstroke, and therefore probably account for non-homogeneous activation of the left ventricle.

Myocardial perfusion scans have shown that leads with the fQRS complex correspond to AMI lesions. The presence of the fQRS complex in inferior, anterior, and lateral wall leads has been reported to have a sensitivity of 82.7%, 72.7%, and 62.9% in diagnosing myocardial infarctions, respectively.[2] In our study, the patients with anterior wall infarction had higher incidence rates of the fQRS complex in the inferior wall leads compared to the other leads. Therefore, leads with the fQRS complex might not always reveal the presence and location of the infarction site. In general, pathological Q waves are associated with transmural infarction, while the fQRS complex is associated with non-transmural infarction or myocardial ischemia. One potential mechanism that could account for this phenomenon is the presence of a wrapped LAD artery, which is defined as a post-reperfusion coronary angiogram that perfuses at least one fourth of the inferior wall of the left ventricle in the right anterior oblique projection.[20] Partial occlusion of a wrapped LAD may cause inferior wall ischemia. In addition, myocardia surrounding the infarction site has been associated with frequent fQRS complexes.[19] Our findings are consistent with previous studies in that the inferior wall leads were associated with a higher incidence of the fQRS complex, suggesting a poor prognosis.[21-23]

Although the prognosis of myocardial infarction has greatly improved due to effective therapies that decrease the size of the infarction and residual ischemia, a significant number of patients still develop heart failure with adverse events even in the absence of recurrent myocardial infarction.[24-27] In general, basic and clinical characteristics including older age, previous myocardial infarction and diabetes contribute to the progression of heart failure and cardiac death. In addition, an increased incidence of the fQRS complex on 12-lead ECG has also been shown to be a valuable predictor of cardiac-linked events including arrhythmia, heart failure and death in patients with CAD.[7,11] However, several previous studies with similar patient populations have failed to demonstrate the predictive value of fQRS.[28,29] In this study, we evaluated various risk factors for all-cause mortality in patients with AMI, and Cox regression analysis showed that the fQRS complex was an independent risk factor for all-cause mortality ($p = 0.040$). This is consistent with a previous meta-analysis which showed that fQRS was well correlated with all-cause mortality.[30]

Brenyo et al.[31] also reported that fQRS complexes located inferiorly were an appropriate predictor of sudden cardiac-linked death/implantable cardioverter defibrillator shock. Korhonen et al.[32] also demonstrated a higher incidence of sudden cardiac death (40%) in post-MI patients. Furthermore, they showed that a higher fragmentation index was more strongly associated with patients who died of heart failure than with those who died of sudden cardiac death. In this study, during a mean follow-up period of 27.8 ± 15.5 months, a significantly higher number of patients with fQRS died compared to those without fQRS. Hence, our findings are consistent with the previous studies, and support the prognostic value of the fQRS complex.

ECG performance has been proposed to be a predictor for late clinical outcomes in patients with MI,[33] and a significantly decreased left ventricular ejection fraction (LVEF) has been reported to be associated with fQRS at admission and at 6 months of follow-up.[34] In addition, fQRS complexes detected on ECG have been reported to reflect the severity of left ventricular dilation and decrease in ejection fraction.[15,35] Consistent with these findings, we found a significant correlation between LVEF and fQRS complex. However, owing to the retrospective study design, we did not include the ejection fraction of the patients in the follow-up study. It has been suggested that continued remodeling of the myocardium after MI, even in patients without Q waves, can eventually promote dilation of the left ventricle, failure and even mortality in patients with fQRS.

This study has some limitations. This was a retrospective study with a relatively small sample size performed at a single center. In addition, patients with ECG suggesting bundle branch block were excluded. However, such patients comprise a significant subset of patients with ACS, and emerging evidence suggests the

presence of the fQRS complex in the ECG of this patient population, and therefore should be included in future studies. In addition, due to the nature of this study, fQRS beyond 48 hours was not analyzed in order to ensure that only early-stage electrocardiograms were included. The sensitivity of fQRS in diagnosing NSTEMI was lower in our study than in a previous report,[2] which can probably be attributed to the small sample size in our investigation.

CONCLUSIONS

In conclusion, our study demonstrated the limited diagnostic value of the fQRS complex in diagnosing NSTEMI within 48 hours of onset. The fQRS complex may serve as a prognostic factor for all-cause mortality in patients with AMI. Further studies with a more robust design and larger sample size are needed to confirm our findings.

DISCLOSURE

The authors declare no conflict of interest.

AUTHORS' CONTRIBUTIONS

LD and ZJY participated in the case collection, data analysis and helped to draft the manuscript. LL participated in the design of the study and performed the statistical analysis. ZWX conceived of the study, and participated in its design and coordination and helped to draft the manuscript. All authors read and approved the final manuscript.

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