Med. J. Cairo Univ., Vol. 93, No. 2, Accepted 12/3/2025
DOI: 10.22608/MJCU. 843-849, June 2025
www.medicaljournalofcairouniversity.net

Evolution of Fragmented QRS in Patients with New-Onset Left Bundle Branch Block Acute Coronary Syndrome Undergoing Primary Percutaneous Coronary Intervention

ISMAIL M. IBRAHIM, M.D.; MANAR M. AL-ZAKI, M.D.; MESBAH T. HASSANEIN, M.D. and HOSSINY E. HOSSINY, M.Sc.

The Department of Cardiology, Faculty of Medicine, Zagazig University

Abstract

Background: The lack of "ST segment resolution sign" after primary PCI represents one of the challenges in diagnosis and management of patients with acute coronary syndrome (ACS) presenting with left bundle branch block (LBBB). The evolution of fragmented QRS (fQRS) in non-LBBB ACS patients was found to be associated with sub-optimal revascularization and poor prognosis.

Aim of Study: This study aimed to assess clinical and angiographic factors associated with the evolution of fQRS in ACS patients presenting with LBBB.

Patients and Methods: This prospectively studied 100 patients with ACS presenting with LBBB and treated with primary PCI. Serial ECGs were obtained over the first 2 days and examined for evolution (group 1) or absence/resolution (group 2) of fQRS. Clinical and angiographic data of groups 1 were analyzed.

Results: Evolution of fQRS occured in 39 (39%) of patients (group 1). In this group, higher percentage of male patients (p=0.004), higher rate of current smoking (p=0.04), higher admission SBP and Killip class (p=0.031 and <0.001; respectively), higher admission HsTnT and CK-MB (p<0.001; for each), lower LVEF at discharge (p<0.001), longer pain-to-door time (p=0.01) and lower MBG (p<0.001) were found in group 1. On regression analysis, presence of MBG 0-1, lower LVEF, higher admission SBP, higher admission CK-MB, and longer pain to door time were found to be independently associated with evolution of QRS.

Conclusions: Evolution of fQRS in ACS patients presenting with LBBB is independently associated with lower LV function and impaired microvascular perfusion. So, fQRS, as asimple marker, may be useful instratification of high-risk patients with increased extent of infarcted myocardium in LBBB ACS.

Correspondence to: Dr. Ismail M. Ibrahim, The Department of Cardiology, Faculty of Medicine, Zagazig University

Key Words: *Acute coronary syndrome – Left bundle branch block – Fragmented QRS – Myocardial blush grade – Microvascular perfusion.*

Introduction

ANATOMICALLY, the anterior half of the left bundle branch is supplied mainly by the first septal perforators of left anterior descending artery, whereas the posterior half is supplied by the right coronary artery **[1]**. Of patients with acute coronary syndrome (ACS), new-onset left bundle branch block (LBBB) occurs in 1-8% **[2]**. Studies on ACS patients presenting with (presumably) new-onset LBBB are conflicting. Some studies reported a higher prevalence of three-vessel and left main disease, cardiogenic shock, pulmonary edema, sudden cardiac death; all-cause mortality **[3-5]**. On the other hand, other studies found no significant association between the presence of LBBB and the severity of coronary artery disease **[6]**. Diagnosis and management of ACS patients presenting with LBBB remain challenging. One challenge is the lack of "ST segment resolution sign", a robust non-invasive marker of microvascular reperfusion **[7]**.

Fragmented QRS complex (fQRS) was originally defined by Das et al. **[8]** as presence of variousr SR patterns or notching of the S or Rwave in the absence of typicalbundle branch block and a QRS duration of <120ms. The presence of fQRS on surface electrocardiography (ECG) was found to reflect a heterogenous depolarization secondary to myocardial edema, fibrosis **[9]** or microvascular dysfunction **[10]**. Similarly, fragmentation of wide QRS with typical BBB patterns was investigated and found to be a sign of myocardial scarring and a predictor of mortality **[11]**. Ozcan et al. **[12]** found that despite complete ST-segment resolution in ST elevation myocardial infarction, fQRS was inde-

pendently associated with impaired microvascular myocardial perfusion.

We aimed to assess clinical and angiographic factors associated with the evolution of fQRS in ACS patients presenting with LBBB.

Patients and Methods

Study population: This prospective, non-randomized observational study was conducted in Cardiology Department, Zagazig University, Egypt during the period from July 2020 through September 2024. It included 100 consecutive patients with suspected ACS and (presumably) new-onset LBBB referred for primary percutaneous coronary intervention (PCI). LBBB was diagnosed by the presence of QRS duration ›_120ms in addition to a QS orrS complex in lead V1 and absence of Q in lead V6 **[13]**. Patients were excluded if they have any of the following: Moderate-severe valvular heart disease, dilated cardiomyopathy, previous coronary revascularization, hyperkalemia, digoxin toxicity; pre-excitation or implanted pacemakers. The study protocol was approved by institution's human research committee and every patient gave informed written consent.

Clinical assessment and laboratory measurements: Hypertension was defined as a systolic blood pressure >140 and/or a diastolic blood pressure >90 during hospitalization or prior use of an antihypertensive drug. Diabetes mellitus (DM) was defined as HbA1C >6.5g/dL or use of oral hypoglycemic agents and/or insulin. Hypercholesterolemia was defined as total cholesterol level >200mg/dL or prior use of statins. Positive family history of premature coronary artery disease (CAD) was defined as presence of CAD in a close relative (men <55 and women <65 years of age). Current smoking was defined as cigarette smoking during the last month. Detailed drug history was taken from each patient. Additionally, admission blood pressure, heart rate, Killip class; and pain to balloon time were recorded for each patient. Baseline high-sensitive troponin T (HsTnT), creatine kinase-myocardial band (CK-MB), complete blood count; and creatinine were obtained on admission. Estimated glomerular filtration rate (eGFR) was calculated.

Surface 12-lead electrocardiogram: Was recorded in every patient to confirm the diagnosis of LBBB and to detect QRS fragmentation. Fragmentation of QRS with LBBB morphology (QRS ›_120 ms) was defined as the presence of >2 notches (at least 1 notch more than the typical BBB) or multiple notches of the R wave or >2 notches in the nadir of the S wave, in 2 contiguous leads corresponding to a major coronary artery territory (Fig. 1) **[11]**. Patients were categorized into two subgroups according to evolution of new-onset of fQRS (Group 1) and absence/resolution of fQRS (Group 2) at 48 hours after primary PCI.

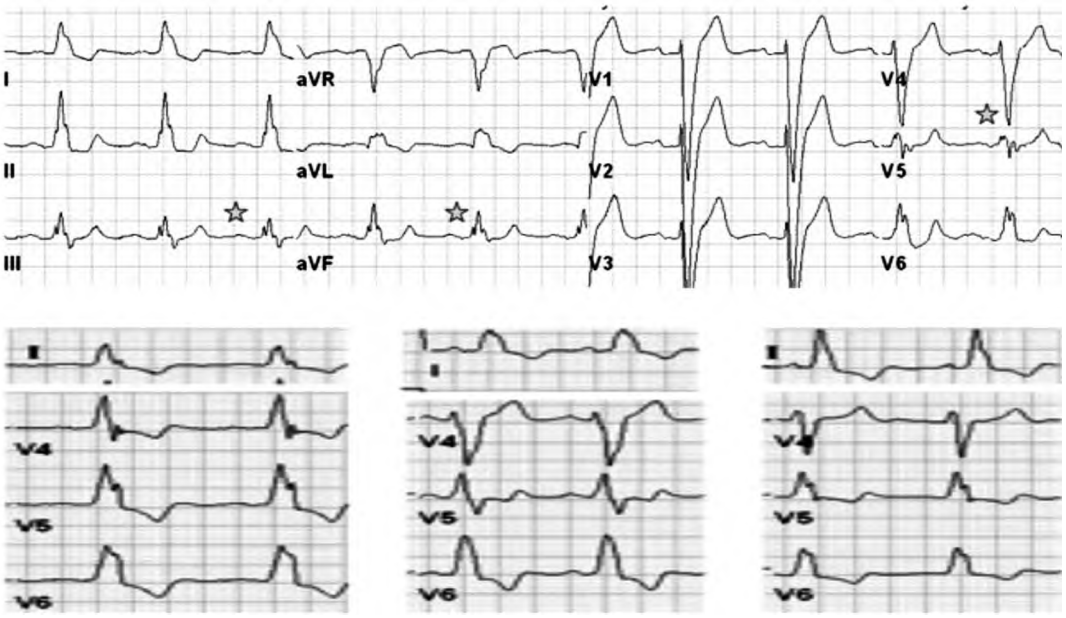

Fig. (1): Examples of fragmented LBBB (A) and non-fragmented LBBB (B). Asterisks denote QRS fragmentation **[10]**.

Primary percutaneous coronary intervention (PCI): All patients underwent primary PCI through femoral approach. PCI to infarct related artery (IRA) was the default strategy with non-culprit artery PCI being done only in presence of cardiogenic shock. The use of devices as well as intra-coronary IIb/IIIa inhibitors were left to the discretion of the operator. Procedural success was defined as a residual stenosis of <20% without major complications viz. stent thrombosis, coronary dissection/perforation, need for emergent coronary artery bypass surgery, no reflowor death. Post-procedural corrected thrombolysis in myocardial infarction (TIMI) frame count (cTFC) and myocardial blush grade (MBG) were measured as previously described **[14,15].**

Statistical analysis: Continuous data were presented as mean ± SD and were compared using the Student's *t*-test (in case of normality) or Mann-Whitney U test (in case of non-normality). Categorical data were presented as frequencies (percentages) and were compared using the Chi-square or Fisher exact test. Binary logistic regression using the enter method was done to determine independent predictors of new-onset or persistence of fQRS in ACS patients presenting with LBBB. A *p*-value of less than 0.05 was considered significant. All tests were performed using SPSS version 20 (SPSS Inc., Chicago, IL).

Results

Comparison between baseline clinical and laboratory characteristics of both groups (Table 1). Percentage of male gender was significantly higher in patients with myocardial infarction presenting with LBBB and fQRS treated with primary PCI (group 1) (64.4% vs 34.4%; p=0.004). Similarly, higher rate of current smoking (p=0.04), higher admission SBP (p=0.031), higher Killip class at admission (p<0.001), higher admission HsTnT (p<0.001), higher admission CK-MB (p<0.001); and lower LVEF at discharge (p<0.001) were found in group 1.

Table (1): Comparison between clinical characteristics of both groups.

	Total (n=100)	Group 1 LBBB+fQRS (n=39)	Group 2 LBBB-fQRS (n=61)	*p*-value
Age (years)	56.1±7.3	57.5±6.2	55.7±5.5	0.082
Male gender, n (%)	46 (46%)	25 (64.1%)	21 (34.4%)	0.004
Hypertension, n (%)	68 (68%)	27 (69.2%)	41 (67.2%)	0.49
Diabetes mellitus, n (%)	51 (51%)	21 (53.8%)	30 (49.2%)	0.30
Current smoking, n (%)	66 (66%)	28 (71.8%)	38 (62.3%)	0.04
Family history of premature CAD	16 (16%)	7 (17.9%)	9 (14.8%)	0.38
Admission SBP (mmHg)	133.9±22.6	137.7±19.4	128.6±19.5	0.031
Admission DBP (mmHg)	83.6±11.0	84.6±9.3	81.8±12.5	0.28
Admission heart rate, beat/minute	79.7 (11.4)	83.2 (16.2)	78.1 (10.4)	0.10
In-hospital Killip class >1, n (%)	21 (21%)	13 (33.3%)	7 (11.5%)	<0.001
Admission HsTnT (ng/L)	1108.4±841.5	1256.6±593.1	902.12±711.1	<0.001
Admission CKMB (U/L)	176 (103–283)	237 (146–346)	137 (75–242)	<0.001
HbA1c (g/dl)	7.4 (1.7)	7.5 (1.9)	6.4 (1.8)	0.13
Baseline eGFR (mL/min/1.73m^2)	95.9±35.7	94.6±35.1	98.6±33.4	0.06
Presence of fQRS on admission	70 (70%)	24 (61.5%)	36 (59.0%)	0.08
LVEF at discharge (%)	45.1±7.2	42.6±5.9	48.8±6.3	<0.001

CAD : Coronary artery disease. fQRS : Fragmented QRS.
CKMB: Creatine kinase-myocardial band. HsTnT: High-sensitive troponin T.
DBP : Diastolic blood pressure. LVEF : Left ventricular ejection fraction.
eGFR : Estimated glomerular filtration rate. SBP : Systolic blood pressure.

Comparison of procedural characteristics of both groups (Table 2). The time from the onset of symptoms to hospital admission (angina-to-door time) was significantly higher in group 1 compared to group 2 (p=0.01). On coronary angiography, both groups showed similar rates of presence of multivessel disease, similar stent number and length. On the contrary, Group 1 showed higher corrected TIMI frame countas well as higher rate of patients with MBG 0-1 (p<0.001 for each).

Table (2): Comparison between procedural characteristics of both groups.

	Total (n=100)	Group 1 LBBB+fQRS (n=39)	Group 2 LBBB-fQRS (n=61)	*p*-value
Pain to door time (minutes)	172.1±87.8	194.5±89.1	151.4±74.6	0.01
Multivessel disease, n (%)	24 (24%)	9 (23.1%)	15 (24.6%)	0.16
Tirofiban administration, n (%)	184 (50.5%)	13 (33.3%)	21 (34.4%)	0.62
No of stents	1.43 (0.44)	1.41 (0.46)	1.27 (0.37)	0.52
Stent length (mm)	26.6±7.6	27.2±8.1	25.5±7.1	0.064
cTFC	21.8 (3.4)	28.1 (4.3)	18.3 (2.8)	<0.001
MBG:				
0-1	57 (57%)	33 (84.6%)	24 (39.3%)	<0.001
2-3	43 (43%)	6 (15.4%)	37 (60.7%)	<0.001

cTFC: Corrected TIMI frame count.
MBG: Myocardial blush grade.
TIMI: Thrombolysis in myocardial infarction.

Regression analysis for prediction of new-onset or persistence of fQRS. Higher admission SBP, higher admission CK-MB, longer pain to door time; and presence of MBG 0-1, lower LVEF, higher admission SBP, higher admission CK-MB, and longer pain to door time were found to be independently associated with evolution of QRS in patients Apresenting with LBBB.

Table (3): Regression analysis for prediction of new-onset or persistence of fQRS.

	Univariate Analysis		Multivariate Analysis	
	OR (95% CI)	*p*	OR (95% CI)	*p*
Male gender	2.67 (1.36-5.24)	0.004	0.65 (0.29-1.43)	0.30
Current smoking	1.37 (1.28–1.44)	0.014	1.24 (1.08–1.34)	0.154
Admission SBP (mmHg)	1.65 (1.48–1.86)	<0.001	1.47 (134–1.62)	0.001
In-hospital Killip class >1	1.006 (1.001-1.01)	0.01	1.001 (0.996-1.007)	0.76
AdmissionHsTnT (ng/L)	1.07 (1.03–1.11)	<0.001	1.04 (0.98–1.10)	0.082
Admission CKMB (U/L)	1.04 (1.0–1.09)	<0.001	1.02 (0.86–1.56)	<0.001
LVEF at discharge (%)	0.78 (0.63–0.95)	<0.001	0.85 (0.67–0.91)	<0.001
Pain to balloon time (minutes)	1.06 (1.01-1.13)	0.01	1.35 (0.996-1.87)	<0.001
MBG 0-1	2.49 (1.91–3.09)	<0.001	1.57 (1.46–1.68)	<0.001

CK-MB: Creatine kinase-myocardial band.
HsTnT : High-sensitive troponin T.
MBG : Myocardial blush grade.
PCI : Percutaneous coronary intervention.
SBP: Systolic blood pressure.

Discussion

The results of our study revealed that the evolution of fragmented QRS in ACS patients presenting with LBBB is independently associated with lower MBG after primary PCI, lower LV function, uncontrolled hypertension, larger infarct sizes as well as late presentation to primary PCI.

MBG, first described by Van't Hof et al. **[15]**, is a simple angiographic observation for assessment of microvascular circulation. During the course of myocardial infarction, occlusion of microvascular bed occurs due to inflammatory edema, vasospasm, distal thromboembolism and reperfusion injury **[16].** Reduced MBG, as well as no-reflow, occurs more frequently with old age, diabetes, high neutrophil count, late presentation as well as large infarcts **[17].** Studies on prognostic value of low MBG revealed that it is associated with poorer LV function as well increased mortality **[18,19].** Classically, ST segment resolution after primary PCI is considered a robust marker of restoration of microvascular circulation **[7].** In ACS patients with LBBB, this sign is lacking adding to the difficulties in the management of these patients.

In the current study, we found that the evolution of fragmented QRS in ACS patients presenting with LBBB is associated with lower MBG. Establishment of good circulation at microvascular level allows rapid and homogenous ventricular depolarization which is represented by lack of QRS fragmentation on surface ECG. High CK-MB levels and

late presentation, well established factors associated with microvascular occlusion, were found in our study as independent predictors for the evolution of fQRS.

Uncontrolled hypertension was found as an independent predictor of fQRS in our study. Eyuboglu M et al. ***[20]*** found that systolic blood pressure is significantly associated with presence of fQRS even in the absence of left ventricular hypertrophy. Fragmented QRS was also found to be associated with reverse dipping, a high-risk feature in hypertensive individuals ***[21]***. Chronicpressure overload leads to excessive accumulation of collagen fibers and connective tissue matrix within the myocardium which results in heterogenous ventricular depolarization ***[22]***. Mahfouz et al. ***[23]*** demonstrated the association between low coronary flow reserve and the presence of fQRS in patients with masked hypertension postulating the presence of microvascular dysfunction as a mechanism.

In our study, the evolution of fQRS was associated with lower LV ejection fraction at discharge. Previous magnetic resonance-based studies revealed the fQRS early post-myocardial correlate to poor LV function and unfavorable LV remodeling ***[24,25]***.

The rate of evolution of fQRS was much higher in male participants in the current study. This finding is in line with previous studies gender differences in prevalence and prognostic value of fQRS ***[26,27]***.

Our study has some limitations. First one is the relatively small number of patients. Second, myocardial perfusion was assessed by angiography. The use of advanced techniques, e.g. magnetic resonance, could have yielded more accurate results. Third one is the lack of data on clinical end points, e.g cardiovascular mortality as well as revascularization. However, a future study is planned to address these limitations.

Conclusions:

In ACS presenting with LBBB, 39% of patients present with QRS fragmentation on surface ECG. The presence of fragmented wide QRS was found to be associated with lower MBG, uncontrolled hypertension at admission, larger infarct sizes as well as late presentation to primary PCI. Hence, fQRS is a simple marker that can be used to risk stratify ACS patients presenting with LBBB.

Conflict of interest: The authors declare that they have no conflict of interest.

References

1- FRINK R.J. and JAMES T.N.: Normal blood supply to the human His bundle and proximal bundle branches. Circulation, 47: 8–18, 1973.

2- NEELAND I.J., KONTOS M.C. and DE LEMOS J.A.: Evolving considerations in the management of patients with left bundle branch block and suspected myocardial infarction. J. Am. Coll Cardiol. Jul.60 (2): 96-105, 2012.

3- ANGHEL L., STĂTESCU C., SASCĂU R.A., et al.: Impact of newly diagnosed left bundle branch block on long-term outcomes in patients with STEMI. Journal of Clinical Medicine, 13 (18): 5479, 2024.

4- KIEHL E.L., MENON V., MANDSAGER K.T., et al.: Effect of left ventricular conduction delay on all-cause and cardiovascular mortality (from the PRECISION trial). Am. J. Cardiol., Oct. 1; 124 (7): 1049-1055, 2019.

5- DI MARCO A., RODRIGUEZ M., CINCA J., et al.: New electrocardiographic algorithm for the diagnosis of acute myocardial infarction in patients with left bundle branch block. J. Am. Heart Assoc., Jul 21; 9 (14): e015573, 2020.

6- ZHU T., CHEN M., HU W., et al.: Clinical characteristics and the severity of coronary atherosclerosis of different subtypes of bundle-branch block. Ann. Noninvasive Electrocardiol., Jan. 27 (1): e12883, 2022.

7- CHAO W., XIAOJIN G., LING Li, et al.: Role of ST-segment resolution alone and in combination with TIMI flow after primary percutaneous coronary intervention for ST-segment–elevation myocardial infarction. Journal of the American Heart Association, Jul. 12 (14), 2023. https://doi.org/10.1161/JAHA.123.029670.

8- DAS M.K., KHAN B., JACOB S., et al.: Significance of a fragmented QRS complex versus a Q wave in patients with coronary artery disease. Circulation, 113: 2495-2501, 2006.

9- MAHENTHIRAN J., KHAN B.R., SAWADA S.G. and DAS M.K.: Fragmented QRS complexes not typical of a bundle branch block: A marker of greater myocardial perfusion tomography abnormalities in coronary artery disease. J. Nucl. Cardiol., 14: 347-353, 2007.

10- ZHANG R., CHEN S., ZHAO Q., et al.: Fragmented QRS complex is a prognostic marker of microvascular reperfusion and changes in LV function occur in patients with ST elevation myocardial infarction who underwent primary percutaneous coronary intervention. Exp. Ther. Med., 13: 3231-3238, 2017.

11- DAS M.K., SURADI H., MASKOUN W., et al.: Fragmented wide QRS on a 12-lead ECG: A sign of myocardial scar and poor prognosis. Circ. Arrhythm Electrophysiol., 1 (4): 258–68, 2008.

12- OZCAN F., TURAK O., CANPOLAT U., et al.: Myocardial tissue perfusion predicts the evolution of fragmented QRS in patients with ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention. Ann. Noninvasive Electrocardiol., Sep. 19 (5): 454-61, 2014.

13- GLIKSON M., NIELSEN J.C., KRONBORG M.B., et al.: ESC Guidelines on cardiac pacing and cardiac resynchronization therapy. Eur. Heart J., 42: 3427–520, 2021.

14- GIBSON C.M., MURPHY S.A., RIZZO M.J., et al.: The relationship between the TIMI frame count and clinical

outcomes after thrombolytic administration. Circulation, 99: 1945-50, 1999.

15- VAN'T HOF A.W.J., LIEM A., SURYAPRANATA H., et al.: Angiographic assessment of myocardial reperfusion in patients treated with primary angioplasty for acute myocardial infarction: Myocardial blush grade. Circulation, 97: 2302-6, 1998.

16- GUPTA S. and GUPTA M.M.: No reflow phenomenon in percutaneous coronary interventions in ST-segment elevation myocardial infarction. Indian Heart J., Jul-Aug. 68 (4): 539-51, 2016.

17- WANG J.W., ZHOUD Z.Q., CHEN Y.D., et al.: A risk score for no reflow in patients with ST-segment elevation myocardial infarction after percutaneous coronary intervention. Clin Cardiol., 38 (4): 208–215, 2015.

18- VAN KRANENBRUG M., MAGRO M. and THIELE H.: Prognostic value of microvascular obstruction and infarct size as measured by CMR in STEMI patients. J. Am. Coll. Cardiol. Imaging., 7: 930–939, 2014.

19- ATTACHAIPANICH T., KAEWBOOT K., ATTACHAIPANICH S., et al.: Prognostic impact of fragmented QRS complex on coronary no-reflow phenomenon and in-hospital mortality in myocardial infarction: A systematic review and meta-analysis. JACC, Apr. 85 (12_Supplement) 1833, 2025.

20- EYUBOGLU M., KARABAG Y., KARAKOYUN S., et al.: Usefulness of fragmented QRS in hypertensive patients in the absence of left ventricular hypertrophy. J. Clin. Hypertens (Greenwich), Sep. 19 (9): 861-865, 2017.

21- EYUBOGLU M., KARABAG Y., KARAKOYUN S. and AKDENIZ B.: The effect of circadian blood pressure pattern on presence of fragmented QRS complexes in hypertensive subjects. J. Am. Soc. Hypertens, Aug. 11 (8): 513-518, 2017.

22- DÍEZ J.: Mechanisms of cardiac fibrosis in hypertension. J. Clin. Hypertens (Greenwich), 9: 546-550, 2007.

23- MAHFOUZ R.A., MESBAH M., GAD M.M., et al.: Relationship between Fragmented QRS and Microvascular Dysfunction in Masked Hypertension. Pulse (Basel), Jun. 24; 10 (1-4): 26-33, 2022.

24- LORGIS L., COCHET A., CHEVALLIER O., et al.: Relationship between fragmented QRS and no-reflow, infarct size, and peri-infarct zone assessed using cardiac magnetic resonance in patients with myocardial infarction. Can J. Cardiol., Feb. 30 (2): 204-10, 2014.

25- CHEW D.S., WILTON S.B., KAVANAGH K., et al.: Fragmented QRS complexes after acute myocardial infarction are independently associated with unfavorable left ventricular remodeling. J. Electrocardiol., Jul-Aug. 51 (4): 607-612, 2018.

26- HAUKILAHTI M.A.E., HOLMSTRÖM L., VÄHÄTALO J., et al.: Gender differences in prevalence and prognostic value of fragmented QRS complex. J. Electrocardiol., Jul-Aug. 61: 1-9, 2020.

27- BOZBEYOĞLU E., YıLDıRıMTÜRK Ö., YAZıCı S. , et al.: Fragmented QRS on admission electrocardiography predicts long-term mortality in patients with non-ST-segment elevation myocardial infarction. Ann. Noninvasive Electrocardiol., 21 (4): 352-357, 2016.

تطور التجزؤ فى مقطع QRS لدى مرضى متلازمة الشريان التاجى الحادة المصحوبة بانسداد الضفيرة اليسرى والذين يخضعون للعلاج عن طريق القسطرة الأولية

الخلفية: ينطوى تشخيص وعلاج مرضى متلازمة الشريان التاجى الحادة المصحوبة بانسداد الضفيرة اليسرى على الكثير من التحديات. أحد هذه التحديات هو غياب نزول قطعة ST فى رسم القلب والذى يمثل علامة مهمة على نجاح القسطرة الأولية. ومن المعروف أن تطورالتجزؤ فى مقطع QRS لدى مرضى متلازمة الشريان التاجى الحادة غير المصحوبة بانسداد الضفيرة اليسرى يرتبط بنتائج غير مثالية بعد القسطرة الأولية.

هدف الدراسة: تقييم العوامل السريرية والتصويرية الوعائية المرتبطة بتطور التجزؤ فى مقطع QRS لدى مرضى متلازمة الشريان التاجى الحادة المصحوبة بانسداد الضفيرة اليسرى وما إذا كان هذا التجزؤ يمثل علامة على عدم نجاح القسطرة الأولية.

المرضى والطرق: أجرينا دراسة على ١٠٠ مريض مصاب بمتلازمة الشريان التاجي الحادة المصحوبة بانسداد الضفيرة اليسرى وتم علاجهم عن طريق القسطرة الأولية. تم الحصول على تخطيط كهربية القلب (ECG) متسلسل خلال اليومين الأولين، وفُحصت تخطيطات القلب لدراسة تطور (المجموعة الأولى) أو عدم تطور أو اختفاء (المجموعة الثانية) التجزؤ فى مقطع QRS.

النتائج: تطور التجزؤ فى مقطع QRS لدى ٣٩ مريضًا حيث لوحظ في هؤلاء المرضى ارتفاع فى نسبة المرضى الذكور ومعدل التدخين، وارتفاع فى ضغط الدم الانقباضى وارتفاع انزيمات القلب وانخفاض فى كفاءة البطين الأيسر، ومدة أطول من الألم حتى دخول المريض إلى المستشفى، وانخفاض فى التروية النسيجية مقاسة بدرجة التلون فى عضلة القلب. وعند تحليل الانحدار، وُجد أن ضعف أو غياب التروية النسيجية وارتفاع ضغط الدم الانقباضى وارتفاع مستوى إنزيم CK–MB عند دخول المريض إلى المستشفى، ومدة أطول من الألم حتى دخول المريض إلى المستشفى، كلها عوامل مرتبطة بتطور التجزؤ فى مقطع QRS.

الاستنتاجات: يرتبط تطورالتجزؤ في مقطع QRS لدى مرضى متلازمة الشريان التاجي الحادة المصحوبة بانسداد الضفيرة اليسرى بشكل مستقل بانخفاض وظيفة البطين الأيسر وضعف تدفق الدم إلى الأوعية الدموية الدقيقة. لذا، قد يكون تطورالتجزؤ فى مقطع QRS كعلامة بسيطة مفيدًا فى تصنيف المرضى المعرضين لزيادة حجم احتشاء عضلة القلب ما يعكس كونهم أكثر خطورة.