Original Article

*Caspian Journal of Internal Medicine 2025 (Winter); 16(1): 83-89*
DOI: 10.22088/cjim.16.1.83

# Assessment of the fragmented QRS relative frequency and its predictive value in patients with pulmonary embolism

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**Received:** 17 May 2023
**Revised:** 31 Oct 2023
**Accepted:** 27 Nov 2023
**Published:** 19 Oct 2024

## *Abstract*

***Background:*** Acute pulmonary embolism can quickly cause hemodynamic collapse and death. Recent studies have shown that different characteristics of electrocardiogram (ECG) can be used to predict the prognosis of patients. This study aimed to investigate the relative frequency of fragmented QRS in the ECG of patients with pulmonary embolism and its prognostic value.

***Methods:*** This study was conducted retrospectively. The files of 106 patients hospitalized with a diagnosis of pulmonary embolism from January 2016 to the end of March 2020 were selected and reviewed. The findings of the ECG, including the ST elevation in V1-V4 leads with and without T invention, right axis deviation, right bundle branch block (RBBB), PR, QRS, QTc intervals, type of treatment (thrombolysis or embolectomy), cardiogenic shock, mortality were collected. Finally, the data were recorded and analyzed in SPSS software Version 16**.**

***Results:*** Hypertension, dyslipidemia, and diabetes mellitus were the most frequent risk factors among the patients. The relative frequency of fragmented QRS, at least in one lead, was 26.2%. The use of thrombolysis, mechanical ventilation, embolectomy, cardiogenic shock, and in-hospital death was significantly higher among patients who had fragmented QRS (P<0.001). CTNI was significantly higher in patients with fragmented QRS (P=0.001). In patients with fragmented QRS large vessels, involvement was significantly higher.

***Conclusion:*** This study showed that the presence of fragmented QRS in the ECG of acute embolism patients has a significant relationship with cardiogenic shock, hospital mortality, and the need for advanced treatment methods such as intubation, embolectomy, and the use of thrombolysis.

***Keywords:*** Acute pulmonary embolism, Fragmented QRS, ECG, Prognosis.

***Citation:***
Dehghani MR, Baradaran M, Rostamzadeh A, et al. Assessment of the fragmented QRS relative frequency and its predictive value in patients with pulmonary embolism. Caspian J Intern Med 2025; 16(1): 83-89.

Pulmonary embolism (PE) occurs due to the pulmonary artery occlusion by thrombosis, tumor, air, or fat. It can manifest as an acute, subacute, or chronic process. The acute form presents immediately, the sub-acute form presents in a couple of days or weeks after primary occlusion, and the chronic one develops slowly and can lead to pulmonary arterial hypertension in the following years. Hemodynamic instability and right-side cardiac failure are important prognostic factors. As a simple and non-invasive method, ECG can detect right ventricle ischemic damage (1, 2). In the United States, the incidence of PE is 1 per 100000 adults (3), based on the US General Surgery Department, annually between 100000-180000 death in hospitalized patients is due to PE. Also, PE is the third most common cause of mortality among cardiovascular patients (4). Age over 60 years, active cancer, chronic lung disease, prior heart failure, a respiratory rate over 30, arterial o2 saturation below 90%, and systolic blood pressure under 100 mmHg can affect the outcome. The majority of suspicious PE patients experience chest pain and shortness of breath.

 Publisher: Babol University of Medical Sciences

As PE presentations are nonspecific, missed diagnosis as coronary artery disease, chronic obstructive pulmonary disease (COPD) exacerbation, or pneumonia could be done instead of PE (5). Among the Iranian population, it is estimated that the incidence of DVT is 914.08-2,780.77 cases per 100,000 people, annually, but because there is no registry system, the exact incidence of PE is not confirmed, yet (6). In elderly patients, PE is more prevalent in males than in females. PE usually occurs on the base of deep vein thrombosis and may have a 25% mortality rate if the patient does not receive treatment (7, 8). ECG is a fast and cost-benefit tool for evaluating every PE patient admitted to the emergency room (9). ECG reveals a wide range of abnormalities. It is totally normal in 10-25% of PE patients. According to prior studies, the existence of heart rate>100/min, S1Q3T3, complete right bundle branch block (RBBB), inverted T waves in V1-V4 leads, ST elevation in aVR, arterial fibrillation has been shown to be accompanied by an increased risk of cardiogenic shock and death (10). A dentate QRS wave (fragmented QRS) is a notch in the R or S wave within a narrow QRS complex setting. It is due to heterogeneous ventricular myocardial depolarization, which can result from ischemia, fibrosis, disturbance in the coronary artery microvascular system, or congenital heart disease (10, 11). FQRS reflects scar and disturbances in myocardial perfusion, which are detected and evaluated by 12 leads and are accompanied by congestive heart failure. Some articles have shown that the FQRS is an independent factor in predicting cardiac events in patients with coronary artery disease (12-14). Das et al.'s study revealed greater sensitivity and less specificity in myocardial scar detection by FQRS than Q wave (14). Even in cardiomyopathy, the presence of FQRS is a significant predictive factor for arrhythmia and future cardiac events (15). Qaddoura et al. proved that ECG as a valuable predictive tool in addition to clinical findings in acute PE patients (16). On the other hand, a recent investigation in Iran has studied 280 patients with acute PE found that the presence of FQRS did not have any relationship with hospital death or adverse events (17). This study was conducted, to evaluate the frequency of EKG-fragmented QRS in patients with the diagnosis of acute embolism and determine its predictive value as a rapid method for patients who are considered suspicious for PE and are referred to the emergency department.

## Methods

In this retrospective study, we evaluated 272 files of hospitalized patients with acute embolism who were admitted to Seyed al-Shohada Hospital in Urmia, between 2016-2021. One hundred patients were excluded due to an unconfirmed diagnosis or incomplete data. Finally, 168 patients were included in study. The study was approved by the ethics committee of the university. Inclusion criteria were acute pulmonary embolism confirmation by CT angiography. Patients with a history of coronary artery disease, congestive heart failure, cardiomyopathies, myocarditis, severe heart valve disease, congenital heart disease, and lung lobectomy were excluded. Blood pressure, heart and respiratory rate, body temperature, oxygen saturation and, patients' clinical presentation including dyspnea, angina, palpitation, hemoptysis, and, syncope were recorded. ECG findings including, ST elevation without T inversion in leads V1-V4, right axis deviation (RBBB), PR interval, QRS and QTc, and also laboratory data including cardiac enzymes (CKMB, CTNI, D-dimer), creatinine levels also were analyzed. Echocardiography was done and tricuspid valve regurgitation, right and left ventricular systolic dysfunction, right ventricle enlargement, ejection fraction (EF), pulmonary artery pressure (PAP), atrial fibrillation (AF) were also evaluated.

Types of treatment (thrombolysis, mechanical ventilation, embolectomy surgery), cardiogenic shock, and mortality up to six months after discharge, as the outcome of disease were evaluated. Cardiogenic shock defined as systolic pressure below 90 mmHg or declined equal to or more than 40 mmHg in blood pressure that lasts 15 minutes or more without arrhythmia or hypovolemia. Based on the European Society of Cardiology guideline, Pulmonary Embolism Severity Index (PESI) was determined for each patient. In this scoring system, one point is assigned for each age over 80, as well as a history of cancer, prior cardiorespiratory disease, heart rate over 110 beats per minute, systolic blood pressure below 100 mmHg, and an oxygen saturation under 90%. A final score of zero means a low risk of mortality within 30 days, while a score of one and above means high risk. Patients were divided into two groups based on the presence or absence of FQRS. All patients were diagnosed using computed tomography (CT) angiography. The standard treatment protocol of our heart center followed for all patients. If a patient presented with a systolic blood pressure below 90 mm Hg, or in cases with severe right ventricular enlargement and a positive troponin, fibrinolytic therapy was initiated. For low-risk patients, we started a standard heparin therapy of 80 unit/Kg intravenous stat following 18 unit/Kg infusion to reach aPTT therapeutic rage of 1.5-2.5 times the control value was started.

**Data processing and Statistical Analysis:** The data was analyzed by SPSS software Version 16. Quantitative data were described by the use of mean and standard deviation.

A comparison of quantitative variables was made using an independent t-test .If the distribution was not normal, the Mann-Whitney test was used. A comparison of qualitative variables was made by using the chi-square test. Kaplan-Meier residual analysis was used to estimate the rates of events during hospitalization and one-year rate. The rate of events between two groups with and without FQRS was compared using the Log Rank test. Cox regression analysis was used to investigate outcomes. The result was reported as the relative risk.

**Ethics statement:** The study was reviewed and approved by the Vice-Chancellery for Research of Urmia University of Medical Sciences. (IR.UMSU.REC.1400.158).

## Results

The current study was conducted on 168 patients, 102(60.7%) were women, the mean age of the patients was 62.7±16.3 years. The BMI of the patients was 30.27±4.92 kg/m$^2$. Women's average BMI was more than men's, and this difference was statistically significant (P=0.012). Hypertension following dyslipidemia and diabetes were the most frequent comorbidities (34.5%, 23.2%, 22.6% respectively). In 49.4% of the patients, there was a history of being bedridden, in 24.4% a history of hospitalization due to surgery, and in 23.2% a history of cardiovascular diseases in the family was reported. According to laboratory tests, D-dimer test was positive in 95.4% and the CTNI in 8.3% of patients. According to clinical symptoms, 95.2% of patients had dyspnea, 72% angina, and 57.1 % with palpitation. More frequently, clot was seen in peripheral vessels (66.7% of patients) than central arteries, more frequently in small vessels. Table 1 shows the relation between echocardiography and hemodynamic parameters and QRS fragmentation.

Patients with fragmented QRS had significantly higher incidence of moderate and severe tricuspid regurgitation, right ventricular systolic dysfunction, moderate to severe right ventricular enlargement, right atrial enlargement, lower LVEF, and atrial fibrillation. Also, higher PAP, higher likelihood of ST depression and T invert in precordial leads and higher incidence of QTC prolongation were seen in patients with fragmented QRS than in patients without fragmented QRS. A positive CTNI was seen in 6(4.8%) of patients in non-fragmented QRS group and in 8(18.8%) of patients in fragmented group. (p=0.010). Table 2 shows the relationship between QRS fragmentation and ECG parameters. The creatinine levels in two groups were not statistically different (P=0.830). There was a higher percentage of large-vessel involvement in fragmented QRS group. The use of thrombolysis, mechanical ventilation, embolectomy, cardiogenic shock, and hospital death were significantly higher in FQRS-positive patients (p<0.005). Table 3 shows the association between QRS fragmentation and adverse events. Large vessel involvement was seen dominantly in the fragmented QRS group, whereas small vessel involvement was dominant in the non-fragmented group (P=0.004) (figure 1) in-hospital death was seen in 8% of patients, and was significantly higher in fragmented QRS group (p<0.001).

**Table 1. The relation between echocardiography and hemodynamic parameters and QRS fragmentation**

| variable | Non-fragmented QRS group n=124 | Fragmented QRS group n=44 | P value |
|---|---|---|---|
| **Systolic blood pressure** | 135.0±18.6 | 133.5±16.4 | 0.629 |
| **Diastolic blood pressure** | 87.7±11.2 | 87.8±14.0 | 0.997 |
| **Heart rate** | 96.1±17.8 | 100.5±17.8 | 0.166 |
| **Respiratory rate** | 24.0±3.2 | 24.6±2.7 | 0.248 |
| **Po2** | 92±3.3 | 91.6±3.7 | 0.469 |
| **Tricuspid regurgitation** | | | |
| **No TR** | 22 (18.3) | 4 (9.1) | <0.001 |
| **Mild TR** | 60 (50%) | 11 (25.0) | |
| **Moderate** | 38 (31.7) | 27 (61.4) | |
| **severe** | 0 (0.0) | 2 (4.5) | |

| variable | Non-fragmented QRS group n=124 | Fragmented QRS group n=44 | P value |
|---|---|---|---|
| **Right ventricular systolic dysfunction** | | | |
| **No dysfunction** | 82 (63.8) | 14 (31.8) | |
| **mild** | 20 (16.7) | 8 (18.2) | <0.001 |
| **moderate** | 18 (15.0) | 22 (50.0) | |
| **Left ventricular dysfunction** | | | |
| **No dysfunction** | 77 (64.2) | 19 (43.2) | |
| **mild** | 28 (23.3) | 14 (31.8) | 0.045 |
| **moderate** | 13 (10.8) | 8 (18.2) | |
| **severe** | 2 (1.7) | 3 (6.8) | |
| **Right ventricular enlargement** | | | |
| **No enlargement** | 74 (61.7) | 10 (22.7) | |
| **mild** | 34 (28.3) | 8 (18.2) | <0.001 |
| **moderate** | 12 (10.0) | 23 (52.3) | |
| **severe** | 0 (0.0) | 2 (4.5) | |
| **Left ventricular ejection fraction** | 50.7±5.8 | 46.0±9.6 | 0.002 |
| **Pulmonary artery pressure** | 49.25±6.4 | 52.25±7.5 | 0.013 |

**Table 2. The relationship between QRS fragmentation and ECG parameters**

| variable | Non-fragmented QRS group n=124 | Fragmented QRS group n=44 | P value |
|---|---|---|---|
| **V1**<br>**ST depression with T inversion** | 17 (13.7) | 21 (47.7) | 0.629 |
| **V2** | | | |
| **ST depression with T inversion** | 16 (12.9) | 8 (18.6) | 0.450 |
| **ST depression without T inversion** | 5 (4) | 3 (7) | |
| **V3** | | | |
| **ST depression with T inversion** | 8 (6.5) | 8 (18.2) | 0.029 |
| **ST depression without T inversion** | 5 (4.0) | 4 (9.1) | |
| **V4**<br>**ST depression with T inversion** | 7 (5.6) | 2 (4.5) | 1.00 |
| **Right bundle branch block** | 0 (0.0) | 23 (52.3) | <0.001 |
| **Right axix deviation** | 9 (7.3) | 18 (40.9) | <0.001 |
| **S1T3Q3** | 12 (9.9) | 11 (25) | 0.021 |
| **Atrial fibrilation** | 8 (6.5) | 10 (22.7) | 0.005 |
| **QTc interval (millisecond)** | 351.45±76.28 | 385.3±48.79 | 0.008 |

**Table 3. The association between QRS fragmentation and adverse events**

| variable | Non-fragmented QRS group n=124 | Fragmented QRS group n=44 | P value |
|---|---|---|---|
| **Fibrinolytic therapy** | 21 (16.9) | 27 (61.4) | <0.001 |
| **mechanical ventilation** | 3 (2.4) | 21 (47.7) | <0.001 |
| **Surgical embolectomy** | 1 (0.8) | 7 (15.9) | <0.001 |
| **Cardiogenic shock** | 1 (0.8) | 11 (25) | <0.001 |
| **death** | 1 (0.8) | 13 (29.5) | <0.001 |

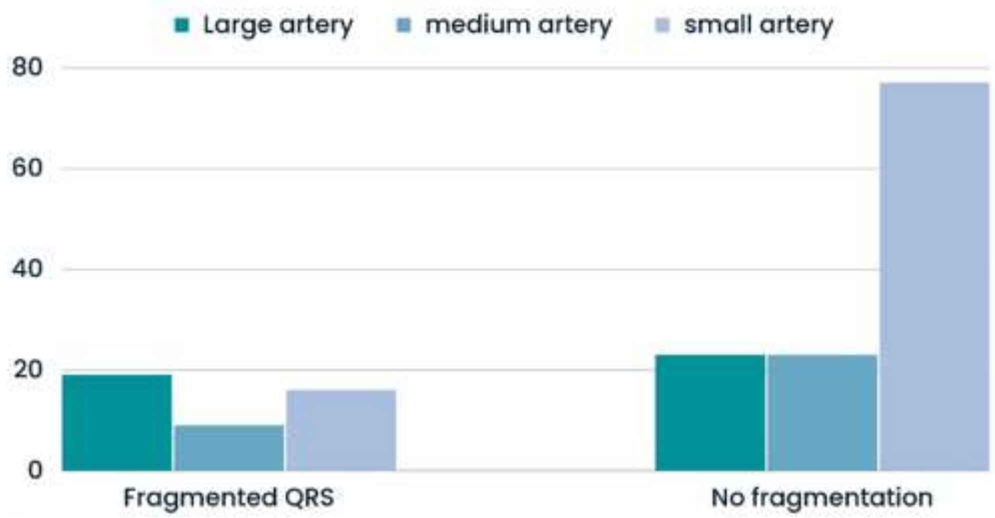


**Figure 1. Pulmonary arteries large vessel involvement was seen dominantly in the fragmented QRS group, whereas small vessel involvement was dominant in the non-fragmented group (P=0.004)**

## Discussion

Acute pulmonary embolism can rapidly lead to hemodynamic collapse and death. Fragmented QRS, can be a valuable predictive factor in patients with pulmonary embolism. ECG is a simple, fast, portable, non-invasive, cheap, and rapidly accessible tool in emergency rooms (18). Fragmented QRS was observed in 26.2% of patients in at least one lead. The most frequent fragmented QRS was in V2, V4, and II, respectively. In the fragmented QRS group, the incidence cardiogenic shock (7.1 %), death during hospitalization (8.3 %), and mortality within six months after the first hospitalization (5.4%) were significantly higher. In Kukla et al.'s study (19), fragmented QRS in lead V1 was observed in 9.8% of patients, which was lower than the results of our study. (16.7% in our study). In another study, 17.14% of the patients with pulmonary embolism had fragmented QRS in their ECG, which was less common compared to our study. Also, the in-hospital mortality rate was 8.7%, which is similar to our findings (17). In Karaca et al.'s study (18) also the study by Kukla et al. (19), in line with the findings of our study, showed that there is a statistically significant correlation between the presence of fragmented QRS in the ECG and the worsening of the clinical outcomes. Also, similar to our study, Cetin et al. (20), showed that the rate of hospital adverse events, in-hospital death, cardiogenic shock, and the use of thrombolytic therapy in fragmented QRS group was significantly higher. The underlying factors resulting in adverse outcome are not described well. Das et al. (21) examined 476 patients with and without a history of coronary artery disease who were referred for a nuclear stress test. They compared the sensitivity and specificity of Q-wave and fragmented QRS for the diagnosis of myocardial injury. They showed that for diagnosing of myocardial scar, fragmented QRS was more sensitive but less specific than Q-wave. Possible mechanisms for the appearance of such fragmentation in patients with pulmonary embolism include a sudden increase in right ventricle filling pressure and a delay in the transmission of electrical impulses. On the other hand, a decrease in the preload of the left ventricle, accompanied by an increase in catecholamine surge, could cause infarction and ischemia of the right ventricle (16). Fragmented QRS in patients with pulmonary embolism may indicate infarction in the full thickness of the right ventricular muscle; which could contribute to the high mortality rate observed in these patients (18). Korhonen et al. also examined 158 patients who had recently experienced myocardial infarction (MI). They suggested that fragmented QRS in patients with MI could predict cardiac death and the progression of heart failure (22). Similarly, other studies have also highlighted the significant value of fragmentation in some cardiovascular diseases such as Brugada syndrome, coronary artery disease, and types of cardiomyopathies (23-25). Contrary to our study, some studies have shown that there is no statistically significant association between the presence of fragmented QRS in the ECG of patients with pulmonary embolism and hospital death, the need for thrombolysis, the need for mechanical ventilation, and embolectomy. The study conducted by Kukla et al. (19), showed that the presence of fragmented QRS in lead V1 had no relationship with hospital mortality.

Also, Zhan et al. found that the presence of fragmented QRS in lead V1 was not related to the need for advanced treatments, inotrope, mechanical ventilation, thrombolysis, and surgery (26). Also, a recent study in Iran has included 280 patients with acute PE. In this study, the incidence of fragmented QRS on ECG of patients was 15% which was lower than our study. (26.1%) The study showed that there was no association between fragmented QRD and hospital mortality, need for fibrinolytic therapy, and other adverse events (17). Our study showed that in Iranian patients, fragmented QRS is an effective tool for predicting adverse outcomes. Limitations of study: This was a single center study, with larger sample size from different regions of Iran the results could be generalized to the general population more accurately. Additionally, subgroup analysis could be done for special population. Due to these controversies more studies with larger sample size could help to reach more precise results. The results of the present study revealed that the presence of fragmented QRS in at least one lead in ECG of patients with acute pulmonary embolism is significantly associated with cardiogenic shock, hospital mortality, and the need for advanced treatment options such as embolectomy, and thrombolytic therapy.

## Acknowledgments

The authors would like to thank Urmia University of Medical Sciences and staff of seyyed al Shohada hospital.

**Funding:** No funding was received to conduct this study.

**Conflict of interests:** All authors declare that there is no possible conflict of interest.

**Authors' contribution:** MR D, MB, AR R, SM conceived, analyzed, and interpreted the results. MR D, AR R, AA, RH conceived the presented idea, supervised the findings of this work, paper drafting and review. All authors approved the contents of the manuscript.

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