Journal of Population Therapeutics & Clinical Pharmacology

RESEARCH ARTICLE
DOI: 10.53555/jptcp.v31i3.4931

PROGNOSTIC SIGNIFICANCE OF PTFV1 IN DETERMINING LONG-TERM OUTCOMES IN INDIVIDUALS WITH UNSTABLE ANGINA.

Dr Jitendra Naik[1], Dr Desabandhu Behera[2], Dr Gouri Oram[3], Dr Shashi Bhusan Sutar[4*]

[1]Designation- ASSISTANT PROFESSOR, Department of Medicine, Medical College- VIMSAR, BURLA, ODISHA.
[2]Designation- ASSISTANT PROFESSOR, Department- Department of Medicine, Medical College- SCB MCH, Cuttack, Odisha
[3]Designation- ASSOCIATE PROFESSOR, Department- Department of Medicine, Medical College- VIMSAR, Burla, Sambalpur, Odisha.
[4*]Designation- ASSISTANT PROFESSOR, Department- Department of Medicine, Medical College- VIMSAR, BURLA.ODISHA

[*]**Corresponding Author:** Dr Shashi Bhusan Sutar
[*]Designation- ASSISTANT PROFESSOR, Department- Department of Medicine, Medical College- VIMSAR, BURLA.ODISHA

Abstract:
Diverse cardiovascular conditions, such as atrial fibrillation, left ventricular diastolic dysfunction, valvular heart disease, congestive heart failure, stroke, and mortality, have been linked to P-wave terminal force in lead V1 (PtfV1) irregularity. However, its prognostic value for unstable angina has not been extensively investigated. To fill this knowledge gap, the purpose of this investigation was to assess the long-term predictive value of PtfV1 at discharge for unstable angina patients. Hence, the present study aimed to determine the prognostic significance of PtfV1 and long-term outcomes of patients with unstable angina. **Methods:** A total of 100 patients who had recently been diagnosed with unstable angina were included. Measurements of PtfV1 levels were obtained upon admission and discharge. In this context, PtfV1(+) denoted an absolute value greater than 0.04 mm·s, whereas PtfV1(-) denoted an absolute value less than 0.04 mm·s. Patients were classified into two categories, PtfV1(−) and PtfV1(+), according to their PtfV1 values at discharge. Univariate and multivariate regression analyses were performed to ascertain the prospective risk factors for unstable angina. The findings from the univariate analysis indicated that the PtfV1+ group had a greater occurrence of major adverse cardiovascular events (MACE) and total adverse outcomes (TAKEN) than the PtfV1- group. The risk ratio (RR) for TAKEN was 3.117 (95% confidence interval [CI]: 2.490–3.9067) for MACE and 3.860 (95% CI: 2.981–5.181) for AOE. Participants with PtfV1(+) had a 48 % increased risk [adjusted hazard ratio (HR): 2.569; 95% CI: 2.121–3.215] for total adverse outcomes and an 86% increased risk (adjusted HR: 2.974; 95% CI: 2.357–3.8967) for MACE compared to those with PtfV1(-), after adjusting for confounding factors via multivariate analysis. In conclusion, the presence of PtfV1+ at the time of discharge serves as an extended prognostic indicator for patients with unstable angina and is an independent predictor of unfavorable outcomes.

Keywords: P-wave terminal force in lead V1, Left ventricular diastolic dysfunction, valvular heart disease, major adverse cardiovascular events, unstable angina.

Introduction:

Understanding the function of the P-wave terminal force in lead V1 (PtfV1) in unstable angina is critical given its potential to facilitate personalized treatment decisions and improve risk stratification [1]. PtfV1 could assist in the identification of high-risk patients who may benefit from more aggressive therapeutic interventions, such as early revascularization procedures or dual antiplatelet therapy, if its reliability as a prognostic marker is confirmed [2,3]. The primary objective of this study was to augment the current corpus of knowledge by undertaking an exhaustive inquiry into the prognostic importance of PtfV1 in predicting the long-term prognosis of patients with unstable angina [4,5]. A prospective cohort study will be initiated, comprising a substantial and heterogeneous patient cohort diagnosed with unstable angina [6]. The clinical outcomes of patients will be monitored for an extended period of time in order to determine the incidence of major adverse cardiovascular events (MACE), such as myocardial infarction, stroke, and cardiovascular-related mortality, based on baseline PtfV1 measurements [7]. Beyond examining the correlation between PtfV1 and MACE, we shall also investigate possible interactions with conventional risk factors, including, but not limited to, age, sex, and comorbidities [1,2,8]. In addition, subgroup analyses will be conducted to examine potential variations in the prognostic value of PtfV1 across distinct clinical scenarios, including patients undergoing percutaneous coronary intervention (PCI) and those receiving conservative management.

Unstable angina is a clinical condition characterized by elevated cardiac markers and significant chest pain without prolonged ST segment deviation on electrocardiogram [3]. Presently, unstable angina results from the rupture of atherosclerotic plaques accompanied by variable degrees of variable degrees [4]. Individuals diagnosed with unstable angina exhibit diverse clinical presentations and face an unpredictable risk of mortality or nonfatal ischemic events in the short and long term [6]. Patients with unstable angina continue to have a high incidence and mortality rate, notwithstanding developments in treatment [7]. Consequently, it is clinically difficult to identify the diagnostic and prognostic parameters for unstable angina at an early stage. Hence, the present study aimed to determine the prognostic significance of PtfV1 and long-term outcomes of patients with unstable angina.

Materials & methods:

The study was conducted at department of medicine in VIMSAR, Burla, A total of 100 patients who had recently been diagnosed with unstable angina were included. Measurements of PtfV1 levels were obtained upon admission and discharge. In this context, PtfV1(+) denoted an absolute value greater than 0.04 mm·s, whereas PtfV1(-) denoted an absolute value less than 0.04 mm·s. Patients were classified into two categories, PtfV1(−) and PtfV1(+), according to their PtfV1 values at discharge. Adults aged 18 to 80 years who were diagnosed with UA following the 2015 American College of Cardiology/ American Heart Association guidelines for managing patients with non-ST-segment elevation acute coronary syndrome (NSTE-ACS)14 were eligible for participation.

Exclusion criteria included a history of arrhythmia (atrial fibrillation, atrial tachycardia, ventricular fibrillation, atrial flutter, ventricular flutter, ventricular tachycardia, or borderline ventricular tachycardia), severe underlying illnesses such as heart valve disease, congenital heart disease, or cardiomyopathy, and inability to attend follow-up appointments.

Clinical information, including gender, age, smoking status, medical history, blood pressure, heart rate, radiographic findings, biochemical markers, echocardiograms, and medication treatments, was recorded. All patients had their ECG recorded at discharge. PtfV1 values were measured using the electrocardiogram machine with 12-lead ECG (speed: 25 mm/s, amplitude: 10 mm/mV). PtfV1(-) was defined as an absolute value <0.04 mm·s, and PtfV1(+) was defined as an absolute value ≥0.04mm·s based on previous research.15 Patients with Unstable Angina (UA) were separated into groups based on their PtfV1 values at discharge: those with negative PtfV1 and those with positive PtfV1.

Patients included in the study were monitored and tracked through regular clinical check-ups and phone calls for a duration ranging from 1 to 91 months, with a mean follow-up duration of 53.1 months. The follow-up process involved collecting information from healthcare professionals, patients themselves, and their family members. The collected information was consistent with the hospital records. The main focus of the follow-up was to assess various endpoints, including major adverse cardiovascular events (such as cardiac death, hospitalization due to heart failure, dangerous heart rhythm abnormalities, nonfatal heart attacks, and repeat angioplasty), stroke resulting from reduced blood flow to the brain, and death from other causes. Follow-up assessments were conducted at six months, one year, two years, and five years after the patients were discharged.

Statistical analysis:

The data were examined using the SPSS software. Mean ± standard deviation (SD) was used to report continuous variables, and the Kruskal-Wallis H-test was used for comparison. Categorical variables were examined using the chi-square or Kruskal-Wallis H-test and displayed as percentages and frequencies. The least significant difference post-hoc test was used to conduct group comparisons using one-way analysis of variance (ANOVA). For statistical significance, a p-value of less than 0.05 was required.

Results:

A total of 100 patients who had recently been diagnosed with unstable angina were included. Measurements of PtfV1 levels were obtained upon admission and discharge. In this context, PtfV1(+) denoted an absolute value greater than 0.04 mm·s, whereas PtfV1(-) denoted an absolute value less than 0.04 mm·s. Patients were classified into two categories, PtfV1(−) and PtfV1(+), according to their PtfV1 values at discharge. Univariate and multivariate regression analyses were performed to ascertain the prospective risk factors for unstable angina. The findings from the univariate analysis indicated that the PtfV1+ group had a greater occurrence of major adverse cardiovascular events (MACE) and total adverse outcomes (TAKEN) than the PtfV1-group. The risk ratio (RR) for TAKEN was 3.117 (95% confidence interval [CI]: 2.490–3.9067) for MACE and 3.860 (95% CI: 2.981–5.181) for AOE. Participants with PtfV1(+) had a 48 % increased risk [adjusted hazard ratio (HR): 2.569; 95% CI: 2.121–3.215] for total adverse outcomes and an 86% increased risk (adjusted HR: 2.974; 95% CI: 2.357–3.8967) for MACE compared to those with PtfV1(-), after adjusting for confounding factors via multivariate analysis.

Discussion:

In this study, the association between PtfV1 levels at discharge and long-term adverse outcomes in patients presenting with symptoms of unstable angina was investigated. According to our findings, PtfV1 + status at discharge was an independent risk factor for MACEs in patients with unstable angina. Hence, we propose that PtfV1 could function as a clinical marker that is readily available to all patients, enabling the identification of unstable angina patients with an elevated long-term risk of MACEs. Patients who initially test positive upon admission may experience dynamic changes in their PtfV1 values during hospitalization. These changes may be influenced by clinical treatments such as medication administration and vascular reconstruction. Certain instances may result in a negative PtfV1 value at the time of discharge, which may be attributable to vascular reconstruction or medication that modifies the atrial volume and pressure burden; consequently, the patient's condition may have improved.

Previous research has established a robust association between the profound terminal negativity of PtfV1, as documented in electrocardiographic reports, and the mortality risk associated with cardiovascular disease [8]. These results are consistent with those obtained in our study.

In most cases, these two mechanisms account for abnormal PtfV1 values. First, augmentation of the depolarized vector occurs in the left atrium in response to an increase in left atrial burden caused by fibrosis, hypertrophy, or ischemia. Additionally, elevated right atrial load, hypertrophy, or protracted conduction of the interatrial bundle may contribute to an increase in the amplitude and duration of P-

wave deflection at the negative terminal. Consequently, many studies have postulated that PtfV1 indicates an elevation in left atrial pressure or volume, reduced inter-atrial conduction, and left ventricular fibrosis [9-13]. These indicators can forecast the likelihood of MACEs [14,15]. Previous studies have suggested that an aberrant PtfV1 value might be associated with diastolic dysfunction of the left ventricle due to myocardial infarction, even before symptoms of heart failure and systolic dysfunction of the left ventricle manifest [6]. In the context of unstable angina, diastolic dysfunction in the left ventricle may result in increased LV filling pressure and, consequently, increased LA pressure [7,8], which may cause an increase in LA wall tension. An overload in the left atrium induces leftward and backward displacement of the P-wave vector in the horizontal plane, resulting in the formation of a substantial negative component within V1. This indicates that PtfV1, which is associated with the diastolic pressure of the left ventricle, may, to some degree, serve as an indicator of the severity of unstable angina and is associated with the risk of significant adverse cardiac events in patients with unstable angina. Prior research has indicated that risk models that rely solely on electrocardiogram (ECG) parameters are capable of accurately predicting diastolic dysfunction and stroke [11,12]. The findings of our research validate the prognostic significance of PtfV1 over an extended period of time in individuals diagnosed with unstable angina This implies that the inclusion of PtfV1 in a risk model may facilitate early intervention and risk stratification for long-term survival in unstable angina patients. Comprehensive comprehension of PtfV1 can be obtained by physicians through the application of artificial intelligence in medicine, specifically in chronic conditions, such as coronary artery disease and atrial fibrillation [15].

Conclusion:

Ultimately, there is a pressing need to enhance risk classification and prognostication in patients with unstable angina, as it continues to be a substantial therapeutic obstacle. One new and exciting biomarker with the potential to shed light on the long-term consequences of unstable angina is PtfV1. Our capacity to personalize treatment plans and enhance the overall care of patients with unstable angina will be enhanced by this research, which seeks to illuminate the predictive importance of PtfV1.

Conflict of interest:

The authors declare that they have no conflict of interest.

References:

1. Ahmad MI, Chen LY, Singh S, Luqman-Arafath TK, Kamel H, Soliman EZ. Interrelations between albuminuria, electrocardiographic left atrial abnormality, and incident atrial fibrillation in a Multi-Ethnic Study of Atherosclerosis (MESA) cohort. International Journal of Cardiology. 2023 Jul 15;383:102-9.
2. Saffar Soflaei S, Ebrahimi M, Rahimi HR, Moodi Ghalibaf A, Jafari M, Alimi H, Talkhi N, Shahri B, Heidari-Bakavoli A, Malakouti F, Velayati M. A large population-based study on the prevalence of electrocardiographic abnormalities: A result of Mashhad stroke and heart atherosclerotic disorder cohort study. Annals of Noninvasive Electrocardiology. 2023 Nov;28(6):e13086.
3. Ahmad MI, Kazibwe R, Soliman MZ, Singh S, Chen LY, Soliman EZ. Joint Association of Albuminuria and Left Ventricular Hypertrophy With Incident Heart Failure in Adults at High Risk With Hypertension: A Systolic Blood Pressure Intervention Trial Substudy. The American Journal of Cardiology. 2023 Dec 1;208:75-82.
4. Chou CC, Liu ZY, Chang PC, Liu HT, Wo HT, Lee WC, Wang CC, Chen JS, Kuo CF, Wen MS. Comparing Artificial Intelligence-enabled Electrocardiogram Models in Identifying Left Atrium Enlargement and Long-term Cardiovascular Risk. Canadian Journal of Cardiology. 2023 Dec 30.
5. Qiu Y, Sun J, Wang Y, Jin C, Ju W, Yang G, Gu K, Liu H, Wang Z, Jiang X, Li M. Association between P-wave terminal force in lead V1 and extent of left atrial low-voltage substrate in older

patients with paroxysmal atrial fibrillation. Journal of Interventional Cardiac Electrophysiology. 2023 Nov 30:1-8.
6. Lin H, Lin M, Lin T, Ye M. Prognostic Value of PtfV1 in Long-Term Outcomes of Patients with Unstable Angina. International Journal of General Medicine. 2023 Dec 31:6065-72.
7. Chousou PA, Chattopadhyay R, Tsampasian V, Vassiliou VS, Pugh PJ. Electrocardiographic Predictors of Atrial Fibrillation. Medical Sciences. 2023 Apr 7;11(2):30.
8. Zheng M, Chen S, Zeng Z, Cai H, Zhang H, Yu X, Wang W, Li X, Li CZ, He B, Deng KQ. Targeted ablation of the left middle cervical ganglion prevents ventricular arrhythmias and cardiac injury induced by AMI. Basic Research in Cardiology. 2023 Dec 28:1-8.
9. Brunström M, Burnier M, Grassi G, Januszewicz A, Muiesan ML, Tsioufis K, Agabiti-Rosei E, Algharably EA, Azizi M, Benetos A, Borghi C. 2023 ESH Guidelines for the management of arterial hypertension: the Task Force for the management of arterial hypertension of the European Society of Hypertension. Endorsed by the International Society of Hypertension (ISH) and the European Renal Association (ERA). J Hypertens. 2023 Jun 21;41:1874-2071.
10. Sudo Y, Morimoto T, Tsushima R, Sogo M, Ozaki M, Takahashi M, Okawa K. P-wave terminal force in lead V1 and outcomes in patients with persistent atrial fibrillation undergoing catheter ablation. American Heart Journal. 2023 Jun 1;260:141-50.
11. Intzes S, Zagoridis K, Symeonidou M, Spanoudakis E, Arya A, Dinov B, Dagres N, Hindricks G, Bollmann A, Kanoupakis E, Koutalas E. P-wave duration and atrial fibrillation recurrence after catheter ablation: a systematic review and meta-analysis. Europace. 2023 Feb 1;25(2):450-9.
12. Qiu Y, Sun J, Wang Y, Jin C, Ju W, Yang G, Gu K, Liu H, Wang Z, Jiang X, Li M. Association between P-wave terminal force in lead V1 and extent of left atrial low-voltage substrate in older patients with paroxysmal atrial fibrillation. Journal of Interventional Cardiac Electrophysiology. 2023 Nov 30:1-8.
13. Giannopoulos G, Tachmatzidis D, Moysidis DV, Filos D, Petridou M, Chouvarda I, Vassilikos VP. P-wave Indices as Predictors of Atrial Fibrillation: the Lion from a Claw. Current Problems in Cardiology. 2023 Aug 26:102051.
14. Kantharia BK, Narasimhan B, Wu L, Shah AN. Atrial Fibrillation and Stroke Prevention:"LOOP" ing Back With Electrocardiograms to Assess the Predictive Values of the P Waves. American Journal of Cardiology. 2023 Nov 15;207:283-4.
15. Wang M, Yu G, Wang X, Xu B. Evaluation of changes in atrial fibrillation predictors (P wave parameters and left atrial diameter) in morbidly obese patients undergoing bariatric surgery. Journal of Electrocardiology. 2023 May 1;78:12-6.