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PREDICTING SUDDEN CARDIAC DEATH IN PATIENTS WITH LEFT VENTRICULAR DYSFUNCTION IS CRITICALLY DEPENDENT ON BNP LEVELS.

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ABSTRACT

Background: Despite major advancements in diagnosis and treatment techniques, sudden cardiac arrest is still a serious health risk. To maximize the advantages of implantable cardioverter-defibrillator (ICD) therapy, it is essential to identify those who are more likely to have sudden cardiac death (SCD). Prohormone (NT-proBNP) is the precursor of BNP (natriuretic peptide) and its amino terminal fragment. Patients who have higher blood levels of these peptides are at a higher risk of experiencing an unplanned cardiac arrest.

Objective: The purpose of this study was to assess the importance of BNP levels in order to predict death from sudden cardiac arrest in patients with left ventricular dysfunction.

Study design: A cross-sectional study

Place and Duration: This study was conducted in Civil Hospital Dow University Karachi from August 2022 to August 2023.

Methodology: All the participants involved in this research had ST elevation myocardial infarction (STEMI), complicated by left ventricular systolic failure. Adult patients with acute myocardial infarction must meet two of the following three criteria: they must have a new or indeterminate left

bundle branch block (LBBB) with ST segment elevation greater than 0.1 mV in limb leads or 0.2 mV in precordial leads, or they must experience the typical chest discomfort behind the breastbone that nitrates can relieve. For five days, a 12-lead ECG was performed every day to guarantee that the chest leads were positioned consistently. Any irregularities found on the ECG were classified according to certain standards.

Results: A total of 80 patients were included in this research, of which 60 were females and 20 were males. The age of the participants ranged from 35 to 80 years. The mean age was 56.8 years. A total of 61 participants underwent primary percutaneous coronary intervention. However, there were 19 patients who got medical therapy. The mean NYHA for survivors was 2.1 after a follow-up clinical examination. A follow-up of 95 days was conducted, out of which 10 (12.5%) of the participants faced life-threatening arrhythmias, 64 (80%) survived, and 6 (7.5%) died because of sudden cardiac arrest.

Conclusion: BNP levels are a significant and independent predictor of sudden death in patients with ischemic cardiomyopathy after an acute myocardial infarction.

Keywords: BNP levels, acute myocardial infarction, sudden cardiac death

INTRODUCTION

Despite major advancements in diagnosis and treatment techniques, sudden cardiac arrest is still a serious health risk [1]. To maximize the advantages of ICD therapy, it is essential to identify those who are more likely to have SCD [2]. The primary metabolism of neurohormones occurs as a result of ventricular strain brought on by high volume and pressure [3]. The brain plays a major role in these [4]. The ones that have been studied the most are prohormone (NT-proBNP), which is the precursor of BNP (natriuretic peptide) and its amino terminal fragment [5].

Previous research has shown that peptides can be used to show how a patient's health is progressing over time, making it easier to figure out how long a patient stays in the hospital and how many die from heart disease [6, 7, 8]. Patients who have higher blood levels of these peptides are at a higher risk of experiencing sudden cardiac arrest. According to the majority of these investigations, left ventricular dysfunction is indicated by noticeably elevated levels of NT-proBNP that are higher than those of BNP, highlighting the predictive sensitivity of NT-proBNP. This emphasizes the possible link between ventricular arrhythmia and B-type natriuretic peptides [9]. Increased wall stress in the left ventricle and systolic or diastolic dysfunction are the causes of the elevation of BNP [10].

The purpose of this study was to assess the importance of BNP levels in order to predict death from sudden cardiac arrest in patients with left ventricular dysfunction.

METHODOLOGY

Left ventricular systolic failure complicated acute STEMI in all of the study participants. There were a total of 80 patients involved in this study.

Adult patients with acute myocardial infarction must meet two of the following three criteria: they must have a new or indeterminate left bundle branch block (LBBB) with ST segment elevation greater than 0.1 mV in limb leads or 0.2 mV in precordial leads, or they must experience the typical chest discomfort behind the breastbone that nitrates can relieve. People had to have a higher cardiac biomarker that showed myocardial injury within the first few hours and show echocardiographic evidence of left ventricular systolic failure (with a left ventricular ejection fraction of 50% or less). Increased creatinine, often known as renal impairment, or systemic sepsis, which results in a nonspecific rise of troponin, are two possible causes of elevated troponin levels. Sample collection must occur before cardiac arrest.

After being told about the procedures, the patient or a close family member gave their consent. The patient's or their family member's complete medical history was acquired. A 12-lead surface

electrocardiogram, a transthoracic echocardiography, a biochemical study, and coronary angiography were all part of the clinical evaluation that took place. An essential component of the clinical evaluation was a comprehensive physical examination.

Clinical scoring systems were used to assess patients who had a heart attack and then developed heart failure. New York Heart Association: This approach assigns a functional status depending on the degree of physical activity and the severity of symptoms. Class I: No limitations on physical activity; moderate exertion does not result in extreme exhaustion, palpitations, or shortness of breath. Class II: Moderate physical activity limitations; tolerable at rest, but frequent exercise causes excessive exhaustion, palpitations, or dyspnea. Class III: Severe limitations on physical activity; comfortable at rest, but extreme exhaustion, palpitations, or dyspnea with little exertion. Class IV: The inability to perform any physical activity without experiencing discomfort; symptoms may intensify with any type of physical activity and may even continue while at rest.

For five days, a 12-lead ECG was performed every day to guarantee that the chest leads were positioned consistently. Any irregularities found on the ECG were classified according to certain standards: ST segment elevation greater than 0.2 mV in precordial leads and more than 0.1 mV in limb leads An erratic heartbeat is known as an arrhythmia. Every patient was placed on their left side and had a transthoracic echocardiographic evaluation (19). All of the exam's images were preserved for future reference, along with the typical ECG. Cardiac biomarkers were assessed at admission. For the purpose of measuring BNP levels, plasma samples were taken from each patient in our study throughout the first 48 hours. All of the patients in our cohort who survived and those who were saved from SCD had extensive follow-up exams, which included evaluations of palpitations and syncopal episodes and a 12-lead ECG to track the development of ventricular arrhythmia within ninety days following the onset of MI.

RESULTS

A total of 80 patients were included in this research, of whom 60 were females and 20 were males. The age of the participants ranged from 35 to 80 years. The mean age was 56.8 years. A total of 61 participants underwent primary percutaneous coronary intervention. However, there were 19 patients who got medical therapy. Table 1 shows the clinical examination of patients. (All the values are in terms of the mean.)

Table No. 1: clinical examination of patients.

Clinical examination	
KILLIP Class	3.2
NYHA	3.2
TIMI risk point	8.4

After a follow-up clinical examination, the mean NYHA for survivors was 2.1. A follow-up of 95 days was conducted, out of which 10 (12.5%) of the participants faced life-threatening arrhythmias, 64 (80%) participants survived, and 6 (7.5%) of the participants died because of sudden cardiac arrest. Table 2 shows the values of the echocardiographic exam taken at the time of admission and after survival. (All values are in terms of the mean.)

Table No. 2: echocardiographic exam taken at the time of admission and after survival

Values at the time of admission	
Regional wall motion abnormalities	14.1
Left ventricular ejection fraction	42.7
Values after survival	
Regional wall motion abnormalities	8.5
Ejection fraction	47.9

Table number 3 shows the cut-offs points for Pro-B-type natriuretic peptides.

Table No. 3: cut-offs points for Pro-B-type natriuretic peptides

Pro-B-type natriuretic peptide	
AUC	70.8%
Cut-off	2.96
P-Value	0.18
Sensitivity	74.1%
Specificity	62.2%

DISCUSSION

One of the main causes of death is acute myocardial infarction (AMI), which continues to have high mortality and morbidity rates even with recent improvements in diagnosis and treatment [11]. Patients with acute ST-elevation myocardial infarction (STEMI) who have elevated BNP levels have been linked to a worse prognosis [12]. For patients who are at a high risk of dying from ventricular arrhythmias, implanted cardioverter-defibrillators (ICDs) are the recommended course of treatment [13]. ICDs are becoming more widely acknowledged as being more beneficial than antiarrhythmic drugs for individuals who have survived cardiac arrest or unstable ventricular tachycardia (VT) [14]. In certain high-risk patients, they seem to be more advantageous than medication treatments for the primary prevention of sudden cardiac death (SCD).

Our goal was to analyze how BNP improved the identification of high-risk individuals who could experience sudden cardiac death (SCD) and improved risk assessment in this patient population. We proved that BNP is a good predictor of SCD in people who have had a ST elevation myocardial infarction (STEMI) and have decreased left ventricular systolic function [15]. This builds on earlier research that showed the marker's usefulness as a prognostic diagnostic in people with acute heart failure. Multiple regression analysis reveals that BNP is an inaccurate risk factor after an acute myocardial infarction (AMI), although it accurately indicates hospitalization duration and fatality rates. Over the course of a year, they evaluated a number of indices, including BNP, in a trial comprising 261 AMI patients in an effort to find important predictors of AMI-related mortality [16]. There are a few, albeit small, studies that link BNP levels to substantial cardiac electrical abnormalities [17, 18].

With respect to BNP's predictive role in ventricular arrhythmias, researchers found that in addition to serving as a marker of left ventricular dysfunction, blood BNP levels also work as predictors of ventricular tachyarrhythmia (VT), with elevated blood BNP levels being associated with a higher likelihood of VT episodes. Numerous investigations have demonstrated links between changes in cardiac or systemic autonomic nerve activity and the onset or development of heart failure, as well as the ventricular arrhythmias that cause potentially fatal cardiac events [19]. Autonomic nerve function contributes to both positive (compensatory) and negative (detrimental) effects on cardiovascular diseases and outcomes.

As is well known, the nervous system controls many of the heart's autonomic processes, including heart rate. However, a number of variables that affect life-threatening cardiac functions collectively include BNP, left ventricular ejection fraction, and coronary risk factors. To put it another way, there isn't a single, trustworthy predictor that can accurately predict the outcome of preventative ICD therapy or arrhythmic mortality. Achieving a more precise risk assessment is essential to optimizing the benefits of ICD treatment, according to Emmanuel G. et al. [20].

CONCLUSION

BNP levels are a significant and independent predictor of sudden death in patients with ischemic cardiomyopathy after an acute myocardial infarction.

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Conflict in the interest
The authors had no conflicts related to their interest in the execution of this study.

Permission
Prior to initiating the study, approval from the ethical committee was obtained to ensure adherence to ethical standards and guidelines.

REFERENCES

1. SULTANA R, MAKWANA D, REHMAN FU, BALOUCH IJ, AZIM W, SOOMRO IE. Prediction of Patient Death due to Sudden Cardiac Arrest with Left Ventricular Dysfunction by Analyzing the Value of BNP: a Prospective Longitudinal Study.
2. MOHAMED AH, YASSIN A, RIZK A, BATTAH A, MOKHTAR M. Evaluation of the Prognostic Value of BNP in Prediction of Sudden Cardiac Death in Patients with Left Ventricular Dysfunction after an Acute MI. The Medical Journal of Cairo University. 2018 Mar 1;86(March):535-41.
3. Yassein A, Hamila M. Usefulness of β-type natriuretic peptide in predicting ventricular arrhythmia in patients with left ventricular dysfunction after an acute myocardial infarction. The Egyptian Journal of Intensive Care and Emergency Medicine. 2021 Dec 1;1(1):15-22.
4. Bettencourt P, Ferreira A, Pereira M, Pardal-Oliveira N, Queir ós C, Ara újo V, Cerqueira-Gomes M, Maciel MJ. Clinical significance of brain natriuretic peptide in patients with postmyocardial infarction. Clinical cardiology. 2000 Dec;23(12):921-7.
5. Richards AM, Nicholls MG, Yandle TG, Ikram H, Espiner EA, Turner JG, Buttimore RC, Lainchbury JG, Elliott JM, Frampton C, Crozier IG. Neuroendocrine prediction of left ventricular function and heart failure after acute myocardial infarction. Heart. 1999 Feb 1;81(2):114-20.
6. Darbar D, Davidson NC, Gillespie N, Choy AM, Lang CC, Shyr Y, McNeil GP, Pringle TH, Struthers AD. Diagnostic value of B-type natriuretic peptide concentrations in patients with acute myocardial infarction. The American journal of cardiology. 1996 Aug 1;78(3):284-7.
7. Kellett J. Prediction of in-hospital mortality by brain natriuretic peptide levels and other independent variables in acutely ill patients with suspected heart disease. The Canadian journal of cardiology. 2004 May 1;20(7):686-90.
8. Wang S, Borah B, Dunlay S, Chamberlain A, Liu J. Sudden Cardiac Death After Revascularization in Patients with Coronary Artery Disease and Left Ventricular Dysfunction. Value in Health. 2018 Sep; 21:S7.
9. Jiang W, Liu Y, He Z, Zhou Y, Wang C, Jiang Z, et al. Prognostic value of left ventricular mechanical dyssynchrony in hypertrophic cardiomyopathy patients with low risk of sudden cardiac death. Nuclear Medicine Communications. 2020; 42(2):182–9.
10. Boldueva S, Leonova IA, Bykova EG, Trostyanetskaya NA. Prognostic value of the left ventricular hypertrophy for sudden cardiac death in patients with myocardial infarction. "Arterial'naya Gipertenziya" ("Arterial Hypertension"). 2009; 15(3):325–9.
11. MALIDZE D. P.2.21 Heart-rate turbulence as risk factor of sudden cardiac death in patients with unstable angina and left ventricular dysfunction. Europace. 2003 Oct; 4:A44.
12. Billman GE. Left ventricular dysfunction and altered autonomic activity: A possible link to sudden cardiac death. Medical Hypotheses. 1986; 20(1):65–77.
13. Angerstein RL, Thompson B, Rasmussen MJ. Preventing Sudden Cardiac Death in Post-Myocardial Infarction Patients with Left Ventricular Dysfunction. The Journal of Cardiovascular Nursing. 2005; 20(6):397–404.

14. Chattopadhyay BP, Ray R, Rath H, Singh S, Chatterjee S. Prognostic value of NT-Pro-BNP in prediction of left ventricular systolic function and outcome of patients of acute coronary syndrome. Indian Heart Journal. 2015 Dec; 67:S23.
15. Pitt B. SUDDEN CARDIAC DEATH: ROLE OF LEFT VENTRICULAR DYSFUNCTION. Annals of the New York Academy of Sciences. 1982 Mar; 382(1 Sudden Corona):218–22.
16. Watanabe J, Shiba N, Shinozaki T, Koseki Y, Karibe A, Komaru T, et al. Prognostic value of plasma brain natriuretic peptide combined with left ventricular dimensions in predicting sudden death of patients with chronic heart failure. Journal of Cardiac Failure. 2005; 11(1):50–5.
17. Waldo AL. Sudden Death in Patients with Myocardial Infarction and Left Ventricular Dysfunction, Heart Failure, or Both. Yearbook of Cardiology. 2006 Jan; 2006:442–3.
18. Bettencourt P, Ferreira A, Dias P, Pimenta J, Friões F, Martins L, et al. Predictors of prognosis in patients with stable mild to moderate heart failure. Journal of Cardiac Failure. 2000 Dec; 6(4):306–13.
19. Berger R, Huelsman M, Strecker K. B-type natriuretic peptide predicts sudden death in patients with chronic heart failure. ACC Current Journal Review. 2002; 11(6):55
20. Wei T, Zeng C, Chen L, Chen Q, Zhao R, Lu G, et al. Systolic and diastolic heart failure are associated with different plasma levels of Btype natriuretic peptide. International Journal of Clinical Practice. 2005; 59(8):891–4.