

Estimation of some vital signs before and after cardiac catheterization for patients with myocardial infarction

Areej Shakeer Jassum¹, Alaauldeen S.M.AL-Sallami^{2*}, Ali Yahya Abdulah alsallami³

¹College of Science, Al-Muthanna University, Iraq. ²Faculty of Science, University of Kufa, Iraq. ³College of Medicine, University of Kufa, Iraq. ¹areejshakeer@mu.edu.iq ²alaaddin.alsallami@uokufa.edu.iq ³aliy.alsalime@uokufa.edu.iq *Corresponding author

Abstract

This study investigated the roles of serum BNP, NGAL, and UPAR in patients with (pre) and (post) catheter myocardial infarction (MI) and compared them with healthy subjects. The included MI patients (n = 50) who underwent catheterization and were admitted to the Cardiac Intensive Care Section of Al-Sadr Teaching Hospital in Al-Najaf Governorate/Iraq had their blood drawn. The patients were divided into two subgroups(pre)and (post) angioplasty. Serum levels of BNP, NGAL, and UPAR were measured in all patients before and after angioplasty. The result of this study showed a significant (p < 0.05) decrease in serum BNP levels in the (pre) patients of MI group compared to the control group (n=25), while serum NAGL and UPAR levels did not show a significant difference (p > 0.05) in the patient's MI group (before) and (after) compared to the control group. In conclusion, serum levels of BNP, NGAL, and UPAR have no potential predictive value in MI patients undergoing catheterization. **Keywords:** Myocardial infarction, Vital signs, Cardiac, Catheterization.

Introduction

One of the most frequently carried out heart operations is cardiac catheterization. Every year, more than 1,000,000 cardiac catheterization operations are carried out in the US (Mozaffarian et al., 2016). The insertion of a tube into a heart chamber or vessel is known as cardiac catheterization (heart cath). commonly carried out in specialized labs equipped with fluoroscopy and highly mobile tables. To increase competence, these "cath labs" are frequently equipped with cabinets of various-sized catheters, stents, balloons, and other medical devices. Fluoroscopic imaging, an electrocardiogram (ECG), and pressure pulses are displayed on screens. (InduSharma,2021). Right heart catheterization,

left heart catheterization, or both can be referred to as cardiac catheterization. Depending on the clinical need, interventional cardiologists can carry out a range of interventions. The procedure can be either diagnostic or therapeutic. Fluoroscopy is typically used to guide and place the catheters during a cardiac catheterization treatment, which is carried out in a cardiac catheterization laboratory. For the operation to be completed safely, the operator needs the assistance of registered nurses and radiologic technologists. Most operations can be carried out using a local anesthetic and minimal to mild sedation, but some will need anesthesia services to provide deep sedation or general anesthesia. the process begins with a comprehensive examination and patient history. The performing interventional cardiologist will select the access for the procedure after defining the clinical issue. These treatments might require venous access, arterial access, or both. The goal of the physical evaluation should be to determine whether the patient is a good candidate for the intended procedure. Reviewing the patient's drug allergies and regular lab work require special care. A complete blood count (CBC) (Al-Sallami et al., 2022), a basic metabolic panel (BMP), a prothrombin time, an electrocardiogram, and a chest Xray are all included in a fundamental workup. Patients who have a known sensitivity to radio-iodinated contrast material will need to take corticosteroids and antihistamines prior to their procedure(Hammood &Al-Sallami,2020). In order to lower the risk of worsening renal function, patients with chronic kidney disease will also require appropriate planning and pre-hydration. (Manda & Baradhi, 2022). A cardiac-derived peptide known as B-type (or brain) natriuretic peptide (BNP) (Saito et al., 1989), although it was first discovered in the porcine brain (Sudoh et al., 1988). The stretching, volume overload, and elevated filling pressure of cardiac myocites cause BNP to be released. All of these activities cause high wall stress, which starts the release of the precursor to BNP. Prior to cleaving into physiologically active BNP and the inactive amino terminal fragment, N-terminal prohormone of BNP, pre-pro-BNP first cleaves into pro-BNP. (Th ygesen et al.,2011; Sudohet al., 2002). Increased plasma BNP levels are observed in myocardial ischemia and left ventricular hypertrophy. (Morita et al., 1993), coronary artery disease (Nishikimi et al.,2004), pulmonary hypertension (Nagaya et al; 1998), and heart failure (Mukoyama et al; 1991), based on the disease's intensity. Because BNP quite better reflects the state of heart failure, it is presently used as a biochemical marker for heart failure in clinical settings. In numerous clinical and epidemiological studies (Sagnella et al., 2001; Groenning et al.,2001; Maisel et al.,2002). It was established that there is a clear correlation between the left ventricle's reduced systolic function and an increase in natriuretic peptides; this allows for potential biochemical diagnosis of heart failure. A 25 kDa glycoprotein of the lipocalin superfamily known as neutrophil gelatinase-associated lipocalin (NGAL) is produced by granulocyte precursors in the bone marrow during a brief window of their development. It is kept in specialized granules of fully developed neutrophils in a gelatinase complex (Kjeldsen et al., 1993). Because its levels rise in the plasma and urine prior to any rise in creatinine levels, NGAL has been viewed in recent

years as primarily an indicator of acute kidney injury (AKI). (Ronco et al., 2007). Additionally, there is growing evidence of increased systemic and myocardial expression of NGAL after an acute MI, supporting the involvement of inflammation in this entity. NGAL has also been linked to cell death, inflammation, and matrix degradation (Malyszko et al., 2008). The urokinase (uPA)-type plasminogen activator system and activation of serine proteases have been shown to play important roles in ventricular and vascular remodeling (Wu Q et al., 2005). uPA is a serine protease that functions in proteolysis during fibrinolysis and in the degradation of the extracellular matrix. Moreover, uPA also mediates signal transduction in cellular adhesion, differentiation, proliferation, and migration via interactions with the G-protein-coupled uPA receptor (uPAR) (Blasi & Carmeliet , 2002). The current study is intended to evaluate this biochemical markers that are prospective for incidence of coronary heart disease in myocardial infarction patients before and after catheterization which may help in early diagnosis.

Materials and Methods

Fifty MI patients were split into two study groups: the control group was made up of 25 men and women who were in good health; the myocardial infarction (MI) patients group before catheterization included 25 subjects; and the myocardial infarction (MI) patients group after catheterization included 25 subjects. The samples were obtained between July and September of 2022 from the Coronary Care Unit (CCU) at Al-Sadder Teaching Hospital in the Iraqi region of Al-Najaf. Patients and the control group varied in age from 35 to 80. Using a needle drained from MI patients before and after catheterization as well as control subjects, five cc of venous blood was collected (Mohy et al., 2022; Alabidi et al., 2023). At room temperature, the blood was allowed to clot in a plain test container. After centrifuging the serum at 3000 rpm for 10 min, it was suctioned, split into aliquots in Eppendorf tubes, and kept at -40 °C. Human BNP, NGAL, and UPAR serum concentrations were measured using an ELISA reagent. The statistical analysis was performed using Graphpad Prism, and the data of the current research were expressed as (Mean Standard Error). When a P-value of 0.05 or higher indicated statistical significance, the contrast between the two groups was examined using the t-test (Albaldawy, 2022).

Results and discussion

The result in figure (1) exhibit significant decrease (p<0.05) in serum levels of BNP in patient (pre) MI group compared with in control group while there is non-significant difference (p>0.05) in serum levels of BNP in patient (post) MI group compared with in control and between (pre) and (post) patient MI group. It may be due to in the identification of impaired left ventricular systolic function. Our findings are in line with those of Groenning et al.(2021), who found a clear correlation between rising BNP levels

and falling LVEF. When BNP and LVEF were correlated, Dilic et al. (2011) discovered a statistically significant negative correlation and a strong association between BNP levels and LVEF. This result may have implications for the use of BNP as a marker for LV dysfunction after AMI. Our current study dis-agree with Richards et al and Talwar et al. (1999) and Talwar et al. (2000). The result in figure (2) exhibit non-significant difference (p>0.05) in serum levels of NGAL in (pre) and (post)patient MI group compared with in control group, also there is non-significant difference in compared between (pre) and (post) patient MI group. Our data agreement with study of Karetnikova et al., showed non- significant NGAL on hospitalization that on day 1, correlated with in-hospital outcomes course during hospitalization for STEMI patient (Karetnikova et al.2017). Previous research and findings suggest that renal dysfunction, not myocardial dysfunction, is the primary factor influencing NGAL levels. According to a recent research that contradicts Hemdahl et al., (2006) MMP-9 co-localized with increased NGAL expression in atherosclerotic plaques in regions with elevated proteolytic activity. Although it is still unknown whether the association between NGAL and inflammation and/or AKI plays a prognostic function, (Lindberg et al., 2012). Shastri et al., (2011) found no correlation between plasma NGAL and LVEF. The result in figure (3) exhibit non-significant difference (p>0.05) in serum levels of UPAR in (pre) and (post)patient MI group compared with in control group, also there is non-significant difference in compared between (pre) and (post) patient MI group. This result agreement with Sorensen et al. The combination of suPAR and troponin did not aid in the diagnosis of acute myocardial infarction (AMI), according to the findings of a trial involving patients with suspected AMI who were brought to the emergency room. (Sorensen et al., 2019). SuPAR was a stable plasma biomarker that predicted all-cause mortality and recurrent MI, according to a research by Lyngbaek et al. (2012) that examined the utility of suPAR in the general (long-term) prediction of repeat MI and mortality in patients with STEMI treated with the PCI procedure. But contrary to what Wlazel et al. (2019) found, the level of soluble urokinase plasminogen activator receptors appears to be a helpful independent biomarker for the early prediction of major adverse cardiac events. The level of the biomarker appears to have greater prognostic than diagnostic potential.



Figure 1: Comparison of serum BNP level between (pre.), (post.) myocardial infarction patient and control group. (*): Statistically significant differences (p<0.05).



Figure 2: Comparison of serum NGAL level between (pre.), (post.) myocardial infarction patient and control group. (*): Statistically significant differences (p<0.05).





Reference

Alabidi, H.M., Farhan, A.M., Salh, N.S. and Aljanaby, A.A.J., 2023. New Azo-Schiff Compounds and Metal Complexes Derived from 2-Naphthol Synthesis, Characterization, Spectrophotometric, and Study of Biological Activity. Current applied science and technology, pp.10-55003.

- Al-Sallami, A.S.M., Al-Shimerty, D.F.H., and Rajab, H.H.(2022). Effect of anticardiolipin (IgG-IgM) and its relationship with the level of white blood cells in women undergoing intracytoplasmic sperm injection (ICSI). AIP Conference ,2386, 020034
- Albaldawy, M.T., Al-Sallami, A.S.M., and Alzeyadi, M.(2022). pSerum HLA-G level as a prognostic marker and its correlation with some important markers for malignant and benign prostate hyperplasia. AIP Conference ,2547, 020002
- Blasi F, Carmeliet P. uPAR. (2002) a versatile signalling orchestrator. Nat Rev Mol Cell Biol . 3: 932-943.
- Dilic M, Nalbantic DA, Arslanagic A, Huskic J, Brdjanovic S, Kulic M, et al. (2011) Biphasic and monophasic pattern of BNP release in acute myocardial infarction. *Coll Antropol.* 35(1):155–159.
- Groenning BA, Nilsson JC, Sondergaard L, Kjaer A, Larsson HB, Hildebrant PR. (2001) Evaluation of impaired left ventricular ejection fraction and increased dimension by multiple neurohumoral plasma concentrations. Eur J Heart Fail.3:699-708.
- Hammood, S.A.,and Al-Sallami, A.S.M.(2020). The role of ctx-ii, dyslipidemia,vitamin d in polycystic ovary syndrome. Indian Journal of Forensic Medicine and Toxicology, 14(2), pp. 2197–2201.
- Hemdahl A, Gabrielsen A, Zhu C, et al. (2006) Expression of neutrophil gelatinaseassociated lipocalin in atherosclerosis and myocardial infarction. Arterioscler Thromb Vasc Biol .26:136–142
- Indu Sharma. (2021) Note on Cardiac Catheterization and its Uses Health Econ Outcome Res Open Access .Vol.7, Issue 12: 204.
- Karetnikova V, Osokina A, Gruzdeva O, Uchasova E, Zykov M, Kalaeva V, et al. (2017) Serum neutrophil gelatinase-associated lipocalin the estimation of hospital prognosis in patients with ST-elevated myocardial infarction. PLoS ONE 12(7): e0180816.
- Kjeldsen L, Johnsen AH, Sengelov H, Borregaard N. (1993) Isolation and primary structure of NGAL, a novel protein associated with human neutrophil gelatinase. J Biol Chem .268:10425–10432.
- Lindberg S, Pedersen SH, Mogelvang R, et al. (2012) Prognostic utility of neutrophil gelatinase-associated lipocalin in predicting mortality and cardiovascular events in patients with ST-segment elevation myocardial infarction treated with primary percutaneous coronary intervention. J Am Coll Cardiol.60:339–345
- Lyngbaek S, Marott JL, Moller DV, Christiansen M, Iversen KK, Clemmensen PM, Eugen-Olsen J. et al. (2011) Usefulness of soluble urokinase plasminogen activator receptor to predict repeat myocardial infarction and mortality in patients

with ST-segment elevation myocardial infarction undergoing primary percutaneous intervention. *Am J Cardiol*.110(12):1756–1763.

- Maisel, A.S.; Krishnaswamy, P.; Nowak, R.M.; McCord, J.; Hollander, J.E.; Duc, P.; Omland, T.; Storrow, A.B.; Abraham, W.T.; Wu, A.H.; et al. (2002) Rapid measurement of B-type natriuretic peptide in the emergency diagnosis of heart failure. N. Engl. J. Med.347, 161–167.
- Malyszko J, Bachorzewska G, Malyszko JS, Pawlak K, Dobrzycki S. (2008) Serum neutrophil gelatinase-associated lipocalin as a marker of renal function in hypertensive and normotensive patients with coronary artery disease. Nephrology.13:153–6.
- Manda YR, Baradhi KM. (2022) Cardiac Catheterization Risks and Complications. [Updated 2022 Jun 11]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing.
- Mohy, A.A., Al-Hadraawy, S.K., ALhadrawi, K.K. and Aljanaby, A.A.J., 2022. Incidence and age distribution of Giardia lamblia infection for sex years in Al-Najaf province in Iraq. Journal of Pharmaceutical Negative Results, pp.1041-1046.
- Morita, E.; Yasue, H.; Yoshimura, M.; Ogawa, H.; Jougasaki, M.; Matsumura, T.; Mukoyama, M.; Nakao, K. (1993) Increased plasma levels of brain natriuretic peptide in patients with acutemyocardial infarction. Circulation 88, 82–91.
- Mozaffarian D, Benjamin EJ, Go AS, Arnett DK, Blaha MJ, Cushman M, Das SR, de Ferranti S, Després JP, Fullerton HJ, Howard VJ, Huffman MD, Isasi CR, Jiménez MC, Judd SE, Kissela BM, Lichtman JH, Lisabeth LD, Liu S, Mackey RH, Magid DJ, McGuire DK, Mohler ER, Moy CS, Muntner P, Mussolino ME, Nasir K, Neumar RW, Nichol G, Palaniappan L, Pandey DK, Reeves MJ, Rodriguez CJ, Rosamond W, Sorlie PD, Stein J, Towfighi A, Turan TN, Virani SS, Woo D, Yeh RW, Turner MB., American Heart Association Statistics Committee. Stroke Statistics Subcommittee. Heart Disease and Stroke Statistics-2016 Update (2016) A Report From the American Heart Association. Circulation. 26;133(4):e38-360.
- Mukoyama, M.; Nakao, K.; Hosoda, K.; Suga, S.; Saito, Y.; Ogawa, Y.; Shirakami, G.; Jougasaki, M.; Obata, K.; Yasue, H.; et al. (1991) Brain natriuretic peptide as a novel cardiac hormone in humans. J. Clin. Investig. 87, 1402–1412.
- Nagaya, N.; Nishikimi, T.; Goto, Y.; Miyao, Y.; Kobayashi, Y.; Morii, I.; Daikoku, S.; Matsumoto, T.; Miyazaki, S.; Matsuoka, H; et al. (1998) Plasma brain natriuretic peptide is a biochemical marker for the prediction of progressive ventricular remodeling after acute myocardial infarction. Am. Heart J.135, 21–28.
- Nishikimi, T.; Mori, Y.; Ishimura, K.; Tadokoro, K.; Yagi, H.; Yabe, A.; Horinaka, S.; Matsuoka, H. (2004) Association of plasma atrial natriuretic peptide, N-terminal proatrial natriuretic peptide, and brain natriuretic peptide levels with coronary

arterystenosis in patients with normal left ventricular systolic function. Am. J. Med.116, 517–523.

- Richards MA, Nicholls MG, Yandle TG, Ikram H, Espiner EA, Turner JG, et al. (1999) Neuroendocrine prediction of left ventricular function and heart failure after acute myocardial infarction. *Heart.* 81:114–120.
- Ronco C. (2007) N-GAL: diagnosing AKI as soon as possible. Crit Care. 11:173.
- Sagnella GA. (2001) Measurement and importance of plasma brain natriuretic peptide and related peptides. Ann Clin Biochem .38:83-93.
- Saito, Y.; Nakao, K.; Itoh, H.; Yamada, T.; Mukoyama, M.; Arai, H.; Hosoda, K.; Shirakami, G.; Suga, S.; Minamino, N.; et al. (1989) Brain natriuretic peptide is a novel cardiac hormone. Biochem. Biophys. Res. Commun.158, 360–368.
- Shrestha K, Borowski A, Troughton R, Thomas J, Klein A, Tang W. (2011) Renal dysfunction is a stronger determinant of systemic neutrophil gelatinase-associated lipocalin levels than myocardial dysfunction in systolic heart failure. J Card Fail.17:472–478
- Sorensen NA, Donmez G, Neumann JT, Nikorowitsch J, Rubsamen N, Blankenberg S, Westermann D. et al. (2019) Diagnostic value of soluble urokinase-type plasminogen activator receptor in addition to high-sensitivity troponin I in early diagnosis of acute myocardial infarction. *Biomolecules*.9(3):108.
- Sudoh, T.; Kangawa, K.; Minamino, N.; Matsuo, H. (1988) A new natriuretic peptide in porcine brain. Nature. 332, 78–81.
- -Talwar S, Squire BI, Downie PF, McCullough AM, Campton MC, Davies JE, et al. (2000) Profile of plasma N-terminal pro-BNP following acute myocardial infarction. *Eur Heart J.* 21:1514–1521.
- Th ygesen K, Mair J, Mueller C, Huber K, Weber M, Plebani M, et al. (2011) Recommendations for the use of natriuretic peptides in acute cardiac care, A position statement from the Study Group on Biomarkers in Cardiology of the ESC Working Group on Acute Cardiac Care. Eur Heart J.
- Wlazel RN, Migala M, Zielinska M, Pawlicki L, Rosniak-Bak K, Szadkowska I. (2019) Soluble urokinase plasminogen activator receptor in one-year prediction of major adverse cardiac events in patients after first myocardial infarction treated with primary percutaneous coronary intervention. *Arch Med Sci*.15(1):72–77.
- Wu Q, Kuo HC, Deng GG. (2005) Serine proteases and cardiac function. Biochim Biophys Acta .1751: 82-94.