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Predictive Value of Combining the Level of Small and Dense Low Density Lipoprotein and N-Terminal Pro-Brain Natriuretic Peptide for Contrast-Induced Acute Kidney Injury in Patients with Acute ST-Segment Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention

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Abstract

Background: To investigate the relationship between the incidence of Contrast-Induced Acute Kidney Injury (CI-AKI) and the level of Small And Dense Low Density Lipoprotein (sd-LDL) and N-Terminal Pro-Brain Natriuretic Peptide (NT-proBNP) in patients with acute ST-Segment Elevation Myocardial Infarction (STEMI) undergoing primary Percutaneous Coronary Intervention (PCI), and to further compare the predictive value of sd-LDL, NT-proBNP and their combination for CI-AKI.

Methods: 415 consecutive STEMI patients who underwent emergency PCI from November 2019 to December 2020 were recruited. Venous blood samples were obtained from all patients before PCI, All patients were divided into groups according to the median of sd-LDL and NT-proBNP to compare the incidence of CI-AKI. Logistic regression analysis was used to study the risk factors of CI-AKI in patients with STEMI after emergency PCI. ROC curve was drawn to evaluate the predictive value of sd-LDL, NT-proBNP and their combined level for CI-AKI.

Results: Patients with high sd-LDL and high NT-proBNP levels had a higher incidence of CI-AKI. The area under the ROC curve was 0.791 (95%CI: 0.736 ~ 0.846 ($p<0.001$), the sensitivity was 84.5%, the specificity was 64.5%, and the sensitivity and specificity were higher than those of sd-LDL or NT-proBNP alone. Multivariate Logistic regression analysis showed that the level of sd-LDL * (OR=1.165, 95% CI: 1.105 ~ 1.228,$p<0.001$), NT-proBNP (OR=2.309, 95%, CI 1.753 ~ 3.043, $p<0.001$), NLR (OR=1.165, 95%, CI 1.101 ~ 1.232, $p<0.001$), eGFR (OR=0.985, 95%, 0.977 ~ 0.993, $p<0.001$) were the independent influencing factor of CI-AKI ($p<0.05$).

Conclusion: High sd-LDL and high NT-proBNP are risk factors for the incidence of CI-AKI, and the combination of them can improve the accuracy of predicting the CI-AKI in STEMI patients undergoing primary PCI.

Keywords: Percutaneous coronary intervention; STEMI; Contrast- induced acute kidney injury; Sd-LDL; NT-proBNP

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Introduction

In recent years, with the rapid progress and continuous development of coronary intervention diagnosis and treatment technology, the widespread use of contrast media has led to an increasing incidence of CI-AKI [1-3]. CI-AKI will significantly prolong the hospitalization time, increase the economic burden and increase the incidence of cardiovascular adverse events. It has been found that compared with ordinary LDL, sd-LDL has the characteristics of small particles, high density, low charge, long half-life, easy oxidative modification, easy to be recognized by macrophages, phagocytosis and so on. Sd-LDL is more atherosclerotic and is an independent risk factor for cardiovascular disease [4,5]. NT-proBNP is the cleavage product of Brain Natriuretic Peptide (BNP) and is a sensitive indicator of ventricular wall tension. Clinically, the plasma levels of BNP and

NT-proBNP are often used as objective markers of early damage to cardiac function [6,7]. This characteristic makes NT-proBNP have a better value in early diagnosis of renal function damage. However, the combination of the sd-LDL and NT-proBNP has not been previously explored, to the best of our knowledge. Therefore, the current prospective study investigated the value of sd-LDL combined with NT-proBNP for predicting the risk of CI-AKI in STEMI patients undergoing primary PCI.

Methods

Study population

Between November 2019 to December 2020, a total of 415 patients with STEMI undergoing primary PCI in Affiliated Hospital of Xuzhou Medical University were enrolled consecutively in the study. All patients were prospectively registered. Low osmotic Nonionic iohexol (Yangzijiang Pharmaceutical Group) was used in all patients during percutaneous coronary intervention, and the osmotic concentration was 800 mOsm/Kg. The definition of preliminary diagnosis of STEMI: a class of acute myocardial infarction with typical ischemic chest pain, lasting for more than 20 minutes, increased serum myocardial necrosis markers and dynamic evolution, and ECG with typical ST-segment elevation. Exclusion criteria: (1) complicated with malignant tumor, liver insufficiency, thyroid dysfunction, immune disease and infectious disease; (2) examination of CT, MRI or other contrast agents 2 weeks before selection; (3) use of nephrotoxic drugs such as aminoglycoside antibiotics and non-steroidal anti-inflammatory drugs within 48 hours before operation or 72 hours after operation. (4) severe chronic heart failure (New York Heart Association (NYHA) grade ≥ Level 3), severe valvular heart disease and hemodynamic instability; (5) previous history of chronic glomerulonephritis, nephrotic syndrome, diabetic nephropathy and chronic renal insufficiency; (6) history of trauma or operation 30 days before operation; (7) incomplete clinical data before and after PCI.

Definition

Definition of CI-AKI: excluding other possible causes of renal damage, the serum creatinine level increased by 0.5 mg/dL (44.2 umol/L) or relative value by 25% over the baseline within 48 to 72 hours after the application of contrast agents. The Estimated Glomerular Filtration Rate (eGFR) was calculated by simplified MDRD formula: eGFR (ml.min-1.173-2)=18Scr (mg/dl)-1.154 × age-0.203 × (0.79 female) [8,9].

Research methods

Venous blood samples were taken to improve laboratory examination before PCI, including blood routine, blood lipids (sd-LDL), biochemistry and precursor of amino terminal brain natriuretic peptide. serum creatinine, cystatin C, urea and uric acid were detected again 48-72 hours after PCI. All patients were treated with hydration by intravenous drip of 0.9%NaCl injection at the rate of 1 ml/ (kg •h) within 12 hours after emergency intervention. After PCI, antiplatelet drugs, β-blockers, angiotensin converting enzyme inhibitors or angiotensin □ receptor antagonists, nitrates, calcium channel blockers, low molecular weight heparin anticoagulants and other coronary heart disease treatment drugs were used according to the clinical condition of the patients, so as to avoid the use of nephrotoxic drugs as far as possible. All patients were divided into low sd-LDL group (sd-LDL<0.83 mmol/l) and high sd-LDL group (sd-LDL ≥ 0.83 mmol/l) according to the median of sd-LDL (0.83mmol). According to the median of NT-proBNP (1686.81 pg/ml), all patients were divided into low NT-proBNP group (NT-proBNP<1686.81 pg/ml) and high NT-proBNP group (NT-proBNP ≥ 1686.81 pg/ml). The patients were further divided into three groups: low risk group (low sd-LDL+ and low NT-proBNP), medium risk group (low sd-LDL+high NT-proBNP or high sd-LDL+low NT-proBNP) and high risk group (high sd-LDL+and high NT-proBNP). The incidence of CI-AKI among different risk grades was compared. According to the changes of creatinine value before and after PCI, all patients were divided into CI-AKI group and non-CI-AKI group, and further compared whether there were statistical differences in clinical data and laboratory data between the two groups. Logistic regression analysis was used to study the influencing factors of CI-AKI in patients with STEMI after emergency PCI. ROC curve was drawn to evaluate the predictive value of sd-LDL, NT-proBNP and their combined level on CI-AKI after PCI. According to the above criteria, patients were divided into CI-AKI group and non-CI-AKI group, further studies on CI-AKI risk factors were conducted.

Statistical analysis

SPSS26.0 statistical software was used to analyze the data. For the measurement data of normal distribution, the continuous variables are shown as mean ± standard deviation ($\bar{x} \pm s$), and the independent sample t-test is used to compare the parameter values between the two groups. The NT-proBNP distribution does not conform to the normal distribution, but the natural logarithm (lnNT-proBNP) accords with the normal distribution. For the measurement data of non-normal distribution, the median and quartile M (Q1, Q3) will be displayed, and the Mann-Whitney U test will be used to compare the non-parameter values between the two groups. The classification variables were expressed as numbers and percentages (%), and were compared by χ 2 test or Fisher exact test. Logistic regression analysis was used to evaluate the risk factors of CI-AKI. ROC curve analysis was used to determine the predictive value of each index for the occurrence of CI-AKI. The p values for interaction were calculated in each subgroup. A 2-sided p value<0.05 was considered as statistically significant.

Results

Comparison of general data of patients

A total of 415 patients were included in this study, including 324 male patients (78.1%) and 91 female patients (21.9%). The average age was 62.70 ± 12.92. There were 174 patients with hypertension (41.9%), 127 patients with glycosuria (30.6%), 178 patients with smoking (42.9%). A total of 71 patients developed CI-AKI after PCI, and the incidence of CI-AKI was 17.10%.

Comparison of the incidence of CI- AKI with different risk levels

The incidence of CI-AKI in low sd-LDL group was 6.7%, and that in high sd-LDL group was 27.5%. There was significant difference between the two groups ($\chi2=31.668$, $p<0.001$). The incidence of CI-AKI in low NT-proBNP group was 7.2%, and that in high NT-proBNP group was 27.1%, and there was significant difference between the two groups ($\chi2=28.802$, $p<0.001$). The incidence of CI-AKI was 5% (5/100) in the low risk group, 8.8% (19/216) in the middle risk group and 47.5% (47/99) in the high risk group, and there was significant difference between the two groups ($\chi2=85.234$, $p<0.001$).

Comparison of basic clinical data between CI-AKI group and non-CI- AKI group

There was a significant difference in the basic clinical data between the two groups in terms of diabetes ($p<0.05$), but there was no significant difference in other general data ($p>0.05$), as shown in **Table 1**.

Variable	non-CI-AKI group (n=344)	CI-AKI group (n=71)	P value
Age (year, $\bar{x}\pm s$)	62.72 ± 12.90	62.61 ± 13.12	0.946
Gender (male/female)	271/73	53/18	0.444
Hypertension [n(%)]	144(14.9)	30(42.3)	0.951
Diabetes [n(%)]	94(27.3)	33(46.3)	<0.001
Smoking [n(%)]	145(42.2)	33(46.5)	0.502
Systolic pressure(mmHg, $\bar{x}\pm s$)	126.78 ± 20.90	127.82 ± 22.56	0.708
Diastolic pressure(mmHg, $\bar{x}\pm s$)	78.31 ± 14.22	79.61 ± 14.87	0.49
Contrast dosage>100 ml, [n (%)]	95(27.6)	16(22.5)	0.379
Contrast medium(ml)	126.8 ± 37.0	120.3 ± 29.1	0.338
Beta-blockers [n(%)]	280(81.4)	60(84.5)	0.535
ACEI/ARB [n(%)]	226(65.7)	51(71.8)	0.318
CCB [n(%)]	40(11.6)	6(8.5)	0.438
Diuretics [n(%)]	158(45.9)	35(49.3)	0.605
Statins [n(%)]	319(92.7)	65(91.5)	0.73
Low molecular weight heparin, [n(%)]	187(54.4)	30(42.3)	0.583
Nitrates [n(%)]	250(72.7)	49(69.0)	0.531

Table 1: Comparison of basic clinical data between CI-AKI group and non-CI-AKI group.

Comparison of laboratory data between CI- AKI group and non-CI-AKI group

Comparing the preoperative laboratory indexes of the two groups, there were significant differences in sd-LDL, lnNT-proBNP, creatinine, eGFR and NLR between the two groups. As shown in **Table 2**.

Variable	non-CI-AKI group(n=344)	CI-AKI group(n=71)	p value
Urea, (mmol/l)	5.74 ± 1.98	5.6 ± 2.08	0.586
Creatinine, (μmol/l)	67.35 ± 15.62	75.96 ± 18.97	<0.001
Cystatin C, (mg/l)	1.06 ± 0.35	1.05 ± 0.4	0.815
Serum uric acid, (μmol/l)	318.97 ± 92.38	307.13 ± 87.43	0.322
eGFR, (ml/min/1.73 m2)	119.72 ± 33.42	104.14 ± 33.78	<0.001
Triglyceride, (mmol/l)	1.2(0.88,1.7)	1.26(0.9,1.92)	0.401
Total cholesterol, (mmol/l)	4.28 ± 0.99	4.26 ± 0.99	0.931
HDL-C, (mmol/l)	0.97(0.82,1.13)	1.01(0.82,1.16)	0.613
LDL-C, (mmol/l)	2.61 ± 0.81	2.6 ± 0.8	0.936
Lp(a), (nmol/l)	210(143.25,339.75)	252(158,359)	0.112
sd-LDL, (mmol/l)	0.78(0.56,1.07)	1.27(0.9,1.63)	<0.001
Total bilirubin, (umol/l)	15.1(10.53,20.55)	15.3(10.1,19.8)	0.961
Direct bilirubin, (umol/l)	5.35(3.8,7.3)	5.5(4.2,7.1)	0.913
Albumin, (g/l)	39.42 ± 5.08	38.67 ± 5.57	0.264
Hs-CRP	4.75(2,15)	4.7(2.5,14.1)	0.896
Monocyte count, (×109/l)	0.5(0.38,0.66)	0.48(0.34,0.64)	0.278
Neutrophils, (×109/l)	6.93(5.27,9.00)	8.36(6.73,10.47)	<0.001
Lymphocyte, (×109/l)	1.10(0.70,1.40)	1.30(1.00,1.90)	<0.001
Red blood cell distribution width, (%)	12.7(12.3,13.48)	12.8(12.2,13.1)	0.31
NLR	4.9(3.17,8.06)	8.35(5.04,11.76)	<0.001
Red blood cell distribution width, (%)	12.7(12.3,13.48)	12.8(12.2,13.1)	0.31
Platelet distribution width, (%)	15.86 ± 1.55	15.81 ± 1.44	0.792
lnNT-proBNP	7.11 ± 1.17	8.09 ± 0.9	<0.001

Table 2: Comparison of preoperative laboratory data between CI-AKI group and non-CI-AKI group.

Logistic regression analysis of risk factors of CI-AKI

Diabetes incidence (assignment: no=0, is=1), sd-LDL* (assignment: measured value×10), NT-proBNP (assignment: natural logarithm value of measured value=lnNT-proBNP), NLR (assignment: measured value), eGFR (assignment: measured value) and other indicators with statistical differences in univariate analysis were included in Logistic regression analysis. The results showed that sd-LDL, lnNT-proBNP and NLR were the risk factors affecting CI-AKI, and eGFR was the protective factor affecting CI-AKI, that is, each additional unit of eGFR. As shown in Table 3.

Variable	Univariate analysis			Multivariate analysis		
	OR	95%CI	p value	OR	95%CI	p value
Diabetes	0.433	0.257-0.731	0.002			
Neutrophils	1.174	1.079-1.278	<0.001			
Mononuclear cell	0.382	0.127-1.147	0.086			
eGFR	0.985	0.977-0.993	0.001	0.988	0.978-0.998	0.019
sd-LDL*	1.165	1.105-1.228	<0.001	1.237	1.157-1.323	<0.001
lnNT-proBNP	2.309	1.753-3.043	<0.001	2.421	1.736-3.378	<0.001
NLR	1.165	1.101-1.232	<0.001	1.124	1.051-1.203	0.001

Table 13: Logistic regression analysis of influencing factors of CI-AKI in STEMI patients after emergency PCI.

The ROC curve of sd-LDL, NT-proBNP and their combination to predict the clinical value of CI-AKI

The AUC of sd-LDL level to predict CI-AKI was 0.741 (95%CI 0.679~0.803, p<0.001), and the best cutoff value was 1.215 mmol/l, the sensitivity was 62.0%, and the specificity was 80.8%. The pre-AUC of NT-proBNP for predicting CI-AKI is 0.728 (95% CI 0.673~0.784, <0.001), and the best cut-off value is 1249.00 pg/ml, the sensitivity is 91.5%, and the specificity is 45.6%. The AUC of sd-LDL combined with NT-proBNP for CI-AKI was 0.791 (95%CI: 0.736-0.846, p<0.001), the best cutoff value was 0.123, the sensitivity was 84.5%, and the specificity was 64.5%. Sd-LDL combines NT-proBNP to generate a prediction probability of the incidence of CI-AKI. Further comparison of the area under the ROC curve of the three groups showed that there was no significant difference in AUC between sd-LDL and NT-proBNP, but there were significant differences in AUC between sd-LDL and combination and between NT-proBNP and combination. As shown in Figure 1.

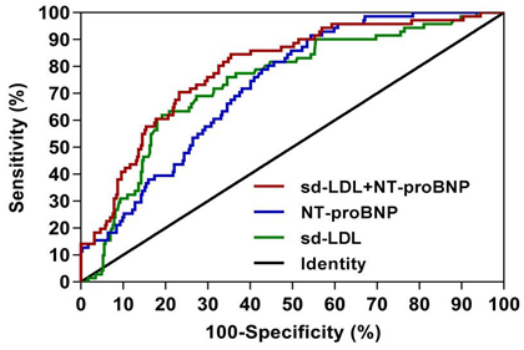

Figure 1: ROC curve of the predictive value of sd-LDL, NT-proBNP and their combination for CI-AKI after PCI.

Discussion

To our knowledge, this is the first study to demonstrate the relationship between the sd-LDL, NT-proBNP and the incidence of CA-AKI in STEMI patients undergoing primary PCI. Our results show that the high level of sd-LDL and NT-proBNP were associated with an increased risk of CA-AKI in STEMI patients undergoing primary PCI. Moreover, the combination of the two can improve the accuracy of predicting CI-AKI after PCI.

Clinical studies have found that compared with ordinary LDL, sd-LDL has the characteristics of low binding to lipoprotein receptor, long half-life, easy oxidative modification, easy to be recognized by macrophages, phagocytosis and so on. With the in-depth development of many studies at home and abroad, the concept of "lipid nephrotoxicity" has been paid more and more attention [10]. Sd-LDL itself contains less antioxidant vitamins and is easy to be oxidized. The modified sd-LDL is not easy to be recognized by lipoprotein receptors on the surface of glomerular epithelial cells and mesangial cells, the binding ability to lipoprotein receptors is decreased, and the time of staying in circulation is prolonged, resulting in lipoprotein (GBM) deposition in mesangial area and glomerular basement membrane and stimulating extracellular matrix (ECM) synthesis [11]. The release of cytokines promotes the proliferation of Mesangial cells and glomerulosclerosis [12]. Studies have shown that after a large number of lipoproteins are ingested by monocytes and macrophages, monocytes and macrophages are transformed into foam cells, and reactive oxygen species, neutral proteases, lysosomal enzymes and other proteases are released into the blood and participate in renal injury. In addition, foam cells produce chemotactic proteins, and more inflammatory cells are chemotactic, attracted, and participate in inflammatory infiltration. The results of this study showed that the incidence of CI-AKI in the high sd-LDL group was significantly higher than that in the low sd-LDL group (27.5% *vs.* 6.7%). Further Logistic regression analysis showed that sd-LDL was an independent risk factor for CI-AKI. The ROC curve of the predictive value of sd-LDL to CI-AKI was drawn, and the results showed that the AUC was 0.741 (95% CI0.679 ~ 0.803, p<0.001). Therefore, patients with STEMI complicated with high level of sd-LDL should be highly vigilant against the occurrence of CI-AKI before PCI [13-16].

NT-proBNP has the characteristics of long half-life, strong stability in vitro, good repeatability and single metabolic pathway, so it is often used to diagnose and evaluate the risk of heart failure and acute coronary syndrome. Studies have shown that NT-proBNP can be used as a biological index to reflect cardiac and renal function. The level of serum NT-proBNP could reflect the changes of renal function better than BNP, and the level of NT-proBNP in patients with renal insufficiency was significantly higher than that in normal subjects. In recent years, it has been reported that NT-proBNP can be used as a predictor of CI-AKI after PCI. The Increase of NT-proBNP synthesis can inhibit myocardial contraction, reduce cardiac output, and then affect renal hemodynamics, leading to CI-AKI. Logistic regression analysis showed that lnNT-proBNP was an independent risk factor for CI-AKI [17-22].

Conclusion

We find that the high levels of sd-LDL and NT-proBNP are risk factors for CI-AKI in STEMI patients undergoing primary PCI. Moreover, the combination of the two can improve the accuracy of predicting CI-AKI after PCI. Further studies are required to determine whether the early treatment of decrease the level of sd-LDL and NT-proBNP improves clinical outcomes in STEMI patients.

Limitation

First, the number of patients enrolled was relatively small. Second, our study failed to follow up patients with CI-AKI to further evaluate the association between sd-LDL and NT-proBNP levels and short or long-term outcomes in patients with contrast agent acute kidney injury. Subsequent studies should further reveal the prognostic value of sd-LDL and NT-proBNP in contrast agent acute kidney injury patients.

Author contributions

YL: methodology, validation, investigation, visualization, writing—original draft preparation; GS: software, formal analysis, data curation; KM: conceptualization, validation, investigation; DZ: investigation; YX: investigation; XZ: investigation; YL: writing—review and editing, funding acquisition; WL: conceptualization, methodology, formal analysis, funding acquisition, project administration, supervision.

Declarations

Conflict of interest

The authors declare that there are no conflicts of interests.

Ethical approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional or national research committee and with the 1964 Helsinki. Declaration and its later amendments or comparable ethical standards. Approval for this study was approved by the ethics committee of the Affiliated Hospital of Xuzhou Medical University, China (ethics approval number: XYFY2018-JS006-01).

Informed consent

Informed written consents were obtained from all patients after providing them a detailed written description of the potential benefits and risks associated with the study.

References

1. Zuo T, Jiang L, Mao S, Liu X, Yin X, et al. (2016) Hyperuricemia and contrast-induced acute kidney injury: A systematic review and meta-analysis. Int J Cardiol 224: 286-294.
2. McCullough PA, Choi JP, Feghali (2016) contrast-induced acute kidney injury. J Am Coll Cardiol. 68: 1465-1473.
3. Mehran R, dangas GD, weisbord SD (2019) Contrast-associated acute kidney injury. N Engl J Med 380: 2146-2155.
4. Fan X, Wang E, Hej X, et al. (2018) Small dense low-density lipoprotein cholesterol and sdldl-C/LDL-C ratio associate with carotid atherosclerotic plaque. Chin J Lab Med 41: 219-226
5. Tsai MY, Steffen BT, Guan W (2014) New automated assay of small dense low-density lipoprotein cholesterol identifies risk of coronary heart disease: the multi-ethnic study of atherosclerosis. Arterioscler Thromb Vasc Biol 34: 196-201.
6. Shimizu N, Kotani K (2020) Point-of-care testing of (N-terminal pro) B-type natriuretic peptide for heart disease patients in home care and ambulatory care settings. Pract Lab Med 22: e00183.
7. Gencer B, Vuilleumier N, Nanchen D, Collet TH, Klingenberg R, et al. (2021) Prognostic value of total testosterone levels in patients with acute coronary syndromes. Eur J Prev Cardiol 28: 235-242.
8. Owen RJ, Hiremath S, Myers A, Fraser-Hill M, Barrett BJ (2014) Canadian Association of Radiologists consensus guidelines for the prevention of contrast-induced nephropathy: update 2012. Can Assoc Radiol J 65: 96-105.
9. Levey AS, Bosch JP, Lewis JB, Greene T, Rogers N, et al. (1999) A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. Modification of diet in renal disease study group. Ann Intern Med 130: 461-470.
10. Hu Z, Ren L, Wang C, Liu B, Song G (2012) Effect of chenodeoxycholic acid on fibrosis, inflammation and oxidative stress in kidney in high-fructose-fed wistar rats. Kidney Blood Press Res 36: 85-97.
11. Gyebi L, Soltani Z, Reisin E (2012) Lipid nephrotoxicity: new concept for an old disease. Curr Hypertens Rep 14: 177-181.
12. Pacher P, Beckman JS, Liaudet L (2007) Nitric oxide and peroxynitrite in health and disease. Physiol Rev 87: 315-424.
13. Lu J, Niu D, Zheng D, Zhang Q, Li W (2018) Predictive value of combining the level of lipoprotein-associated phospholipase A2 and antithrombin III for acute coronary syndrome risk. Biomed Rep 9: 517-522.
14. Wang LJ, Wu MS, Hsu HJ (2012)The relationship between psychological factors, inflammation, and nutrition in patients with chronic renal failure undergoing hemodialysis. Int J Psychiatry Med 44: 105-118.
15. Abbasi A, Corpeleijn E, Gansevoort RT (2013) Role of HDL cholesterol and estimates of HDL particle composition in future development of type 2 diabetes in the general population: the PREVEND study. J Clin Endocrinol Metab 98: E1352-E1359.
16. Bansal SK, Yadav R (2016) A Study of the Extended Lipid Profile including Oxidized LDL, Small Dense LDL, Lipoprotein (a) and Apolipoproteins in the Assessment of Cardiovascular Risk in Hypothyroid Patients. J Clin Diagn Res 10: BC04-BC8.
17. Chae MS, Koo JM, Park CS (2016) Predictive Role of Intraoperative Serum Brain Natriuretic Peptide for Early Allograft Dysfunction in Living Donor Liver Transplantation. Ann Transplant 21: 538-549.

18. Scotti AV, Tura BR, Rocha RG, Albuquerque DC. (2012) Prognostic value of B-type natriuretic peptide in the mortality of patients with acute coronary syndrome. Arq Bras Cardiol 99: 605-612.

19. Li X, Liu C, Mao Z, Qi S, Song R, et al. (2020) Brain natriuretic peptide for predicting contrast-nnduced acute kidney injury in patients with acute coronary syndrome undergoing coronary angiography: A systematic review and meta-analysis. J Interv Cardiol 2020: 1035089.

20. Liu YH, Liu Y, Zhou YL (2015) Association of N-terminal pro-B-type natriuretic peptide with contrast-induced nephropathy and long-term outcomes in patients with chronic kidney disease and relative preserved left ventricular function. Medicine (Baltimore) 94: e358.

21. Lin KY, Wu ZY, You ZB (2018) Pre-procedural N-terminal pro-B type natriuretic peptide predicts contrast-induced acute kidney Injury and long-term outcome in elderly patients after elective percutaneous coronary intervention. Int Heart J 59: 926-934.

22. Goussot S, Mousson C, Guenancia C (2015) N-terminal fragment of pro B-type natriuretic peptide as a marker of contrast-induced nephropathy after primary percutaneous coronary intervention for ST segment elevation myocardial infarction. Am J Cardiol 116: 865-871.