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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Impact of serum alkaline phosphatase level on the pathophysiologic mechanism of contrast-induced nephropathy

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Abstract

Background: Contrast-induced nephropathy (CIN) accounts for 10% of all causes of hospital-acquired renal failure. It leads to a prolonged in-hospital stay, and represents a powerful predictor of poor early and late outcomes. More than half of cases are observed after cardiovascular procedures.

Aim: To determine the predictive value of the serum alkaline phosphatase (ALP) level in the development of CIN, something which has not been assessed before.

Methods: We prospectively evaluated a total of 430 patients with acute coronary syndrome. Patients were classified according to the development of CIN and both groups were compared statistically according to clinical, laboratory and demographic features, including the serum ALP level.

Results: CIN was observed in 20.5% of patients. Advanced age, male gender, elevated creatinine, uric acid and phosphate levels, and low glomerular filtration rate were correlated with the development of CIN. Correlation analysis also showed a significant association between the ALP level and the development of CIN (126.1 ± 144.9 vs. 97.2 ± 46.9, p = 0.004). Univariate regression analysis also showed the impact of ALP on the development of CIN (OR 1.004, 95% CI 1.001–1.007, p = 0.02).

Conclusions: Our outcomes indicate a possible active role of ALP in the mechanism of CIN. An elevated ALP level may predict the development of CIN.

Key words: alkaline phosphatase, contrast-induced nephropathy, pathophysiology

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INTRODUCTION

Contrast-induced nephropathy (CIN) is defined as acute renal damage that develops secondarily to the administration of intravenous contrast agents in interventions performed for diagnostic and therapeutic purposes [1]. More than half of cases are diagnosed after cardiac catheterisation; these cases constitute 10–15% of hospitalisations because of acute renal failure [2]. Pre-procedural creatinine levels and glomerular filtration rate (GFR) have been clearly demonstrated to be predictors of CIN development. These parameters are assessed in the clinical setting, predominantly due to easy accessibility and lower cost, although superior markers are available, including cystatin C and neutrophil gelatinase associated lipocalin (NGAL) [3, 4]. Other simple laboratory predictors, for instance uric acid levels, are also under investigation to define novel determinants of CIN development [5–7]. Serum alkaline phosphatase (ALP) levels have been shown to have a promoting effect on vascular calcification [8–10], and a correlation of ALP with chronic renal failure is also well-established. It has been clearly shown that predominantly bone-associated ALP levels are increased in renal failure secondary to bone destruction, which promotes renal osteodystrophy [11, 12].

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However, the impact of ALP in acute renal failure and CIN has not been reported before. Herein, we aimed to determine the predictive role of the serum ALP level in the development of CIN. We hypothesised that an elevated ALP level may predict CIN development by the mechanism of endothelial dysfunction linked to microvascular calcification.

METHODS

Study population

Our study was conducted at a single regional cardiovascular centre with high patient turnover using a cross-sectional, prospective and observational design. All the patients were informed about the aims of the study and gave their consent. Between 1 October 2009 and 21 October 2011, a total of 430 patients with acute coronary syndrome who had undergone a coronary intervention procedure were enrolled in this study. The exclusion criteria, which may affect the serum ALP level, were as follows: chronic liver disease, active hepatitis, acute and chronic biliary system disease, active infection, chronic inflammatory disease involving the skeletal system, decompensated heart failure, chronic renal disease (GFR < 30 mL/min or haemodialysis treatment) and history of cancer. Patients with reduced renal function (GFR 30–60 mL/min) were hydrated with 0.9% saline at 1 mL/kg/h for 12 h before and after catheterisation. For emergency coronary interventional procedures, physiological (0.9%) saline was given intravenously at a rate of 1 mL/kg/h for 12 h after contrast exposure. In patients with a left ventricular ejection fraction (LVEF) < 40% or overt heart failure, the hydration rate was reduced to 0.5 mL/kg/h. A non-ionic, low-osmolality contrast agent (most frequently Iohexol-Omnipaque®, rarely Iopamidol-Iopamiro® and Iopromide-Ultravist®, very rarely Iodinaxol-Visipaque®) was used in our catheterisation laboratory. Maximal contrast agent usage was limited to 4 mL/kg. In patients with LVEF < 40% and GFR < 60 mL/min, contrast agent usage was limited to 2 mL/kg. In most patients, 100–150 mL of contrast agent was used for each procedure.

Study protocols and definitions

Upon admission, patients were evaluated with anamnesis and physical examination, and then blood samples were taken for analysis. Echocardiography was performed after coronary intervention. Serum creatinine concentrations were measured before and within 48–72 h of contrast agent administration in every patient, and further measurements were performed in all patients who developed CIN. Patient demographics, clinical and angiographic data were assessed. Renal function was assessed by the estimated GFR (eGFR) using the Modification of Diet in Renal Disease Study (MDRD). The MDRD formula is (186 × serum creatinine – 1.154 × age – 0.203) [× 0.742 if female] × [1.212 if black]. CIN was defined as an elevation of serum creatinine by > 0.5 mg/day or > 25% occurring within three days after the intravascular administration of contrast medium, without another aetiology. Serum ALP analysis was performed using a Cobas Integra 400 plus automated enzymatic analyser (Roche Diagnostic, Mannheim, Germany) with 40–129 U/L as the normal range for men and 35–104 U/L for women.

Statistical analysis

Statistical analyses were performed using SPSS 15.0 software (SPSS Inc., Chicago, IL, USA). Mean ± standard deviation, median and [maximum–minimum] were used for continuous variables, while percentages were used for categorical variables. Normal distributions were tested with the one-sample Kolmogorov-Smirnov test. Unpaired t-tests were used to test the differences between the continuous variants, which showed a normal distribution between patient and control groups. Two-group non-parametric comparisons were calculated by the Mann-Whitney U-test when data was non-normally distributed. Pearson χ^2 and Fisher's exact tests were used to test the categorical variants. Univariate and multivariate regression analyses were performed to determine the independent predictors of CIN. The baseline variables for which evident significance ($p < 0.10$) was found by univariate analysis were included in the multivariate logistic regression analysis. The results of the model were reported as a 95% confidence interval (CI) and p-values. All p-values were two-sided in the tests and p-values less than 0.05 were considered to be statistically significant.

This study was approved by the Local Ethics Committee of the hospital.

RESULTS

The baseline demographics and clinical characteristics of the patients are set out in Table 1. The mean age was 64.2 ± 11.2 years, ranging from 22 to 91 years; 270 (62.8%) of the patients were male. The majority of patients had an anamnesis of hypertension and diabetes (74% and 40.1%). The mean ALP level was 103.5 ± 80.0 mg/dL and an elevated ALP level was observed in 80 (22.2%) patients. CIN was observed in 20.5% of patients. Advanced age, male gender, high initial creatinine, uric acid and phosphate levels, and a low GFR as well as a high ALP level, were correlated with the development of CIN (Table 2). Advanced age, male gender, the presence of diabetes, high initial creatinine, uric acid and phosphate levels, a low GFR and a high ALP level were found to be predictors of CIN development in univariate regression analysis (Table 3). However, none of the mentioned clinical and laboratory variables had a significant independent predictive value in multivariate binary logistic regression analysis (Table 3). This result may be explained by the multifactorial pathophysiology of CIN development. A similar impact of each variable may have contributed to the multivariate regression analysis data.

Table 1. Baseline demographic characteristics, clinical features, laboratory results of the patients

Variables	Per cent (n = 430)
Age [years] (range)	64.2 ± 11.2 (22–91)
Sex:	
Male	62.8% (270)
Female	37.2% (160)
Presence of DM	40.1% (171)
Presence of hypertension	74.0% (313)
Presence of dyslipidaemia	49.5% (211)
Smoking	72.7% (312)
Initial creatinine [mg/dL]	1.07 ± 0.27
Third day creatinine [mg/dL]	1.20 ± 0.47
Initial urea [mg/dL]	45.1 ± 19.1
Third day urea [mg/dL]	52.8 ± 29.0
GFR [mL/min]	67.8 ± 21.6
Uric acid [mg/dL]	6.27 ± 2.12
Calcium [mg/dL]	9.18 ± 1.21
Phosphate [mg/dL]	3.52 ± 0.87
ALP [mg/dL]	103.5 ± 80.0
Elevated ALP level	22.2% (80)
Ejection fraction [%]	47.9 ± 14.9
NT-proBNP [mg/dL]	431 ± 2581
CIN development	20.5% (88)

ALP — alkaline phosphatase; CIN — contrast-induced nephropathy; DM — diabetes mellitus; GFR — glomerular filtration rate; NT-proBNP — N terminal pro B-type natriuretic peptide

DISCUSSION

Serum ALP is a membrane-anchored ectoenzyme that catalyses the hydrolysis of organic pyrophosphate, which has been shown to be a protective factor for vascular integrity [11]. An elevated ALP level is commonly reported with renal osteodystrophy [12]. In addition, it has been proposed that ALP may play a role in the pathophysiology of renal dysfunction [13–15]. However, the role of ALP in CIN development is not clear. Herein, we show that ALP may take part in the pathophysiology of CIN development and may be considered a predictor of CIN development. This study could be a pivotal report in the literature, as it evaluated the correlation between serum ALP levels and CIN. Patients with CIN had relatively higher ALP levels with statistical significance.

Contrast-induced nephropathy is one of the most important clinical complications associated with coronary diagnostic and interventional procedures, accounting for 10% of all causes of hospital-acquired renal failure. Its development is associated with increased morbidity and mortality, including the need for transient dialysis and/or extended hospitalisation, and can lead to chronic end-stage renal disease [16].

Many factors play a role in the development of CIN, such as diabetes mellitus, hypertension, age, amount and choice of contrast media, urgent interventions, patient dehydration, and the drugs used [17]. Indeed, there are several traditional and novel markers including creatinine, GFR, cystatin C and NGAL that have been applied by clinicians to assess the individual risk for CIN development following percutaneous coronary intervention. However, there is still debate regarding the predisposing factors for CIN [3, 4]. The complete mechanism of CIN has not been clearly established. In addition to the directly toxic effects of the contrast agent, disturbances in renal blood flow, vasoconstriction of renal vessels, oxidative stress, free radical damage, and endothelial dysfunction are thought to be major mechanisms in the development of CIN [18–20].

Serum ALP, which catalyses the hydrolysis of organic pyrophosphate, has been shown to be a promoting factor in the pathophysiology of vascular calcification through the pyrophosphate pathway [11, 20–22]. The vasculopathic effect of ALP was initially shown in haemodialysis patients [23]. Vascular calcification is one of the major contributors to atherosclerosis, which leads to vascular hardening, ageing and final significant vascular events [24]. Upregulation of serum ALP levels has been observed in vessels with medial calcification, which supports the mediator role of ALP [24]. The serum ALP level has been established as a marker of renal dysfunction in several clinical settings, despite its indefinite role in CIN development [11, 12]. Leibovitch et al. [25] proposed serum ALP as an indicator of renal damage, and CIN itself may also lead to increased ALP levels. Although the impact of ALP on CIN development is not clear, several studies have reported that the urinary ALP level may predict early detection of diabetic nephropathy, even with a normal serum creatinine level [14–16]. Catalytic activities of ALP in the cortical segment have also been proposed as another mechanism of renal dysfunction, especially in transplant patients [13]. Tissue non-specific ALP is expressed in the S1, S2, and S3 segments of the renal tubule [15]. The catalytic activity of ALP leads to the inactivation of inorganic pyrophosphate, which is a potent inhibitor of hydroxyapatite crystal growth and a potential local and circulating inhibitor of vascular calcification. The final result is chondrogenic conversion of vascular smooth muscle cells and vascular mineralisation, which may be one of the mechanisms of renal impairment [11]. Thus, the serum ALP level may predict subclinical renal damage as well as CIN development. In addition, the vasculopathic effect of elevated serum phosphate may also be linked to the catalytic activity of ALP, since phosphate is an end-product of pyrophosphate hydrolysis [11]. Our findings also support this hypothesis, as both correlation analysis and univariate regression analysis showed a correlation between the serum phosphate level and CIN development.

Table 2. Distribution of clinical and demographic characteristics of the patients according to the development of contrast-induced nephropathy

Variables	CIN (+)	CIN (–)	P
Age [years]	66.2 ± 9.9	63.6 ± 11.4	0.05
Sex:			**0.09**
Male	70.5% (62)	60.8% (208)	
Female	29.5% (26)	39.2% (134)	
Presence of DM	47.7% (42)	38.2% (129)	**0.10**
Presence of hypertension	75.6% (65)	73.6% (248)	0.83
Presence of dyslipidaemia	46.5% (40)	50.3% (171)	0.53
Smoking	72.7% (64)	72.7% (248)	1.00
Initial creatinine [mg/dL]	1.13 ± 0.25	1.05 ± 0.27	0.01
Third day creatinine [mg/dL]	1.69 ± 0.70	1.07 ± 0.27	< 0.001
Initial urea [mg/dL]	47.7 ± 19.2	44.4 ± 19.1	0.14
Third day urea	75.8 ± 43.7	46.6 ± 19.5	< 0.001
GFR [mL/min]	64.2 ± 22.6	68.7 ± 21.3	0.08
Uric acid [mg/dL]	6.83 ± 1.9	6.08 ± 2.1	0.02
Calcium [mg/dL]	9.1 ± 1.2	9.2 ± 1.2	0.44
Phosphate [mg/dL]	3.7 ± 1.1	3.4 ± 0.7	0.03
ALP [mg/dL]	126.1 ± 144.9	97.2 ± 46.9	**0.004**
Elevated ALP level	25.0% (20)	21.4% (60)	0.45
Ejection fraction [%]	46.5 ± 13.6	48.2 ± 15.2	0.39
NT-proBNP [mg/dL]	669 ± 3719	351 ± 2067	0.41
Total	20.5% (88)	79.5% (342)	

P < 0.05 is indicated as significant; abbreviations as in Table 1

Table 3. Univariate and multivariate logistic regression analysis to reveal the determinants of contrast-induced nephropathy (CIN)

Dependant variable: CIN	Univariate analysis		Multivariate analysis	
	95% CI	P	95% CI	P
Age [years]	1.000–1.044	0.05	0.945–1.021	0.37
Sex (male/female)	0.392–1.081	0.09	0.192–1.396	0.51
Presence of diabetes mellitus	0.422–1.084	0.10		
Presence of hypertension	0.557–1.645	0.87		
Initial creatinine	1.188–6.504	0.01	0.056–36.50	0.82
Initial urea	0.997–1.020	0.14		
Glomerular filtration rate	0.978–1.001	0.08	0.942:1.040	0.67
Alkaline phosphatase	1.001–1.007	**0.02**	0.998–1.007	**0.28**
Uric acid	1.019–1.356	0.02	0.883–1.251	0.57
Calcium	0.780–1.116	0.44		
Phosphate	1.014–1.805	0.04	0.729–1.581	0.71
NT-proBNP	0.999–1.001	0.69		
Ejection fraction	0.976–1.009	0.39		

P < 0.05 is indicated as significant; CI — confidence interval; NT-proBNP — N terminal pro B-type natriuretic peptide

A major negative criticism about our investigation would be the statistical non-significant multivariate regression analysis regarding ALP levels. One explanation could be the balanced dispersion of the variables in the study sample. Eight clinical and laboratory variables were linked to CIN development in univariate regression analysis, but none of these showed individual significance in multivariate regression analysis. The multifactorial mechanism of CIN has already been defined, including the clinical and laboratory variables assessed in the current study. We believe that balanced dispersion and a similar effect of the variables may have led to the non-significant multivariate regression results. Further investigations with larger samples may clarify the unique impact of each variable in CIN development.

Nonetheless, we should not neglect the impact of ALP in CIN development according to multivariate regression data. The serum ALP level seems to be a powerful predictor of CIN development, and further clinical investigation may confirm this correlation.

Limitations of the study

There are several limitations regarding our study. First of all, it was a non-randomised study performed at a single centre with a relatively small sample size, which necessitates further investigations. In addition, other inflammatory and neurohormonal mediators including C-reactive protein, erythropoietin, nitric oxide, other proinflammatory cytokines and markers of oxidative stress were not measured; these mediators have the potential to affect renal blood flow and may trigger renal medullar ischaemia. Novel markers, including cystatin C and NGAL, which have been described as powerful markers of CIN development, were not evaluated. Herein, we only evaluated the traditional markers of CIN and we characterised serum ALP level as an additional variable. Finally, we evaluated tissue non-specific ALP levels and we did not assess the sub-groups of ALP isoenzymes, including the bone and intestinal isoenzymes. However, the majority of the investigations regarding a linkage between ALP and vascular calcification have been performed using tissue non-specific ALP, which supports our methods.

CONCLUSIONS

Serum ALP is a widely available and inexpensive vasculopathic marker which may predict vascular calcification. Our outcomes indicate a possible active role of ALP in the mechanism of CIN. An elevated ALP level may predict the development of CIN.

Conflict of interest: *none declared*

References

1. Sudarsky D, Nikolsky E. Contrast-induced nephropathy in interventional cardiology. Int J Nephrol Renovasc Dis, 2011; 4: 85–99.
2. Nough H, Eghbal F, Soltani M et al. Incidence and main determinants of contrast-induced nephropathy following coronary angiography or subsequent balloon angioplasty. Cardiorenal Med, 2013 ;3: 128–135.
3. Alharazy SM, Kong N, Saidin R et al. Serum neutrophil gelatinase-associated lipocalin and cystatin c are early biomarkers of contrast-induced nephropathy after coronary angiography in patients with chronic kidney disease. Angiology, 2014; 65: 436–442.
4. Vanmassenhove J, Vanholder R, Nagler E, Van Biesen W. Urinary and serum biomarkers for the diagnosis of acute kidney injury: an in-depth review of the literature. Nephrol Dial Transplant, 2013; 28: 254–273.
5. Demirkol S, Balta S, Akgul EO et al. Hyperuricemia may be related to contrast-induced nephropathy after percutaneous coronary intervention. Clinics (Sao Paulo), 2013; 68: 1071.
6. Boyacioglu M, Turgut H, Akgullu C et al. The efficient of L-carnitine on oxidative stress responses of experimental contrast-induced nephropathy in rat. J Vet Med Sci, 2014; 76: 1–8.
7. Huang SS, Huang PH, Leu HB et al. Association of central pulse pressure with contrast-induced nephropathy and clinical outcomes in patients undergoing coronary intervention. J Hypertens, 2013; 31: 2187–2194.
8. Wannamethee SG, Sattar N, Papcosta O et al. Alkaline phosphatase, serum phosphate, and incident cardiovascular disease and total mortality in older men. Arterioscler Thromb Vasc Biol, 2013; 33: 1070–1076.
9. Park JB, Kang DY, Yang HM et al. Serum alkaline phosphatase is a predictor of mortality, myocardial infarction, or stent thrombosis after implantation of coronary drug-eluting stent. Eur Heart J, 2013; 34: 920–931.
10. Tonelli M, Curhan G, Pfeffer M et al. Relation between alkaline phosphatase, serum phosphate, and allcause or cardiovascular mortality. Circulation, 2009; 120: 1784–1792.
11. Schoppet M, Shanahan CM. Role for alkaline phosphatase as an inducer of vascular calcification in renal failure? Kidney Int, 2008; 73: 989–991.
12. Magnusson P, Sharp CA, Magnusson M et al. Effect of chronic renal failure on bone turnover and bone alkaline phosphatase isoforms. Kidney Int, 2001; 60: 257–265.
13. Schmid H, Mall A, Bockhorn H. Catalytic activities of alkaline phosphatase and N-acetyl-beta-D-glucosaminidase in human cortical nephron segments: heterogeneous changes in acute renal failure and acute rejection following kidney allotransplantation. J Clin Chem Clin Biochem, 1986; 24: 961–970.
14. De Carvalho JA, Piva SJ, Hausen BS et al. Assessment of urinary gamma-glutamyltransferase and alkaline phosphatase for diagnosis of diabetic nephropathy. Clin Chim Acta, 2011; 412: 1407–1411.
15. Nuyts GD, Yaqoob M, Nouwen EJ et al. Human urinary intestinal alkaline phosphatase as an indicator of S3-segment-specific alterations in incipient diabetic nephropathy. Nephrol Dial Transplant, 1994; 9: 377–381.
16. Levy EM, Viscoli CM, Horwitz RI. The effect of acute renal failure on mortality. a cohort analysis. JAMA, 1996; 275: 1489–1494.
17. Deray G. Nephrotoxicity of contrast media. Nephrol Dial Transplant, 1999; 14: 2602–2260.
18. Katholi RE, Woods WT Jr, Taylor GJ et al. Oxygen free radicals and contrast nephropathy. Am J Kidney Dis, 1998; 32: 64–71.
19. Weisberg LS, Kurnik PB, Kurnik BR. Radiocontrast-induced nephropathy in humans: role of renal vasoconstriction. Kidney Int, 1992; 41: 1408–1415.
20. Cantley LG, Spokes K, Clark B et al. Role of endothelin and prostaglandins in radiocontrast-induced renal artery constriction. Kidney Int, 1993; 44: 1217–1223.
21. Johnson RC, Leopold JA, Loscalzo J. Vascular calcification: pathobiological mechanisms and clinical implications. Circ Res, 2006; 99: 1044–1059.
22. Regidor DL, Kovesdy CP, Mehrotra R et al. Serum alkaline phosphatase predicts mortality among maintenance hemodialysis patients. J Am Soc Nephrol, 2008; 19: 2193–2203.
23. Detrano R, Guerci AD, Carr JJ et al. Coronary calcium as a predictor of coronary events in four racial or ethnic groups. N Engl J Med, 2008; 358: 1336–1345.
24. Shanahan CM, Cary NR, Salisbury JR et al. Medial localization of mineralization-regulating proteins in association with Monckeberg's sclerosis: evidence for smooth muscle cell-mediated vascular calcification. Circulation, 1999; 100: 2168–2176.
25. Leibovitch I, Ben-Chaim J, Ramon J, Goldwasser B. Increased serum alkaline phosphatase activity: a possible indicator of renal damage. J Clin Lab Anal, 1991; 5: 406–409.

Wpływ stężenia fosfatazy zasadowej w surowicy na patofizjologiczny mechanizm nefropatii kontrastowej

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Streszczenie

Wstęp: Nefropatia kontrastowa (CIN) stanowi 10% wszystkich jatrogennych niewydolności nerek. Jest ona przyczyną przedłużonej hospitalizacji i stanowi silny czynnik prognostyczny wczesnych i odległych powikłań. Ponad połowa przypadków CIN występuje po procedurach w obrębie układu sercowo-naczyniowego.

Cel: Celem badania było określenie wartości prognostycznej stężenia w surowicy fosfatazy zasadowej (ALP) w odniesieniu do ryzyka rozwoju CIN; zagadnienie to nie było wcześniej oceniane.

Metody: Autorzy dokonali prospektywnej oceny 430 chorych z ostrym zespołem wieńcowym. Chorych podzielono na grupy w zależności od rozwoju CIN, a następnie przeprowadzono statystyczną analizę porównawczą obu grup pod względem parametrów klinicznych, laboratoryjnych i demograficznych, w tym stężenia ALP w surowicy.

Wyniki: U 20,5% chorych stwierdzono CIN. Czynnikami związanymi z rozwojem CIN były: podeszły wiek, płeć męska, podwyższone stężenie kwasu moczowego i ALP oraz niska filtracja kłębuszkowa. W analizie korelacji wykazano istotny związek między stężeniem ALP a rozwojem CIN (126,1 ± 144,9 *vs.* 97,2 ± 46,9; p = 0,004). Również analiza wieloczynnikowa wykazała wpływ stężenia ALP na rozwój CIN (OR 1,004; 95% CI 1,001–1,007; p = 0,02).

Wnioski: Uzyskane wyniki wskazują, że ALP może odgrywać aktywną rolę w mechanizmie rozwoju CIN. Zwiększone stężenie ALP może być czynnikiem prognostycznym rozwoju CIN.

Słowa kluczowe: fosfataza zasadowa, nefropatia kontrastowa, patofizjologia

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