อุบัติการณ์และปัจจัยสัมพันธ์การเกิดภาวะไตวายเฉียบพลันจากสารทึบรังสีในผู้ป่วยภาวะกล้ามเนื้อหัวใจขาดเลือดเฉียบพลันชนิดเอสทียกภายหลังการตรวจสวนหัวใจหรือขยายหลอดเลือดหัวใจแบบฉุกเฉิน

Incidence and Associated factors with Contrast-Induced Nephropathy after Emergency Percutaneous Coronary Intervention in Patients with ST-segment Elevation Myocardial Infarction

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บทคัดย่อ

การศึกษานี้มีวัตถุประสงค์เพื่อค้นหาอุบัติการณ์และปัจจัยสัมพันธ์การเกิดภาวะไตวายเฉียบพลันจากสารทึบรังสีในผู้ป่วยภาวะกล้ามเนื้อหัวใจขาดเลือดเฉียบพลันชนิดเอสทียก ภายหลังได้รับการตรวจรักษาด้วยการตรวจสวนหัวใจหรือขยายหลอดเลือดหัวใจแบบฉุกเฉินในประชากรไทย โดยมีวิธีการดำเนินการวิจัยแบบศึกษาย้อนหลังในผู้ป่วยจำนวน 280 ราย โดยวัดการเพิ่มของระดับครีเอตินินที่เพิ่มขึ้นมากกว่าร้อยละ 25 หรือมากกว่า 0.5 มิลลิกรัม/เดซิลิตร จากค่าพื้นฐานของผู้ป่วย ภายใน 48-72 ชั่วโมงภายหลังได้รับสารทึบรังสีทางหลอดเลือด วิเคราะห์ข้อมูลโดยใช้สถิติพรรณนาและการวิเคราะห์ถดถอยเชิงพหุเพื่อทำนายปัจจัยสัมพันธ์ภาวะไตวายเฉียบพลันจากสารทึบรังสี ผลการวิจัยพบว่าเกิดอุบัติการณ์ร้อยละ 16.4 และพบปัจจัยสัมพันธ์การเกิดภาวะไตวายเฉียบพลันได้แก่ ปริมาณสารทึบรังสีที่ได้รับมากกว่า 160 มิลลิลิตร (AOR 4.66, 95%CI: 2.02-10.71, p<.001); ภาวะหัวใจล้มเหลว (AOR 4.43, 95%CI: 1.80-10.93, p = .001); โรคความดันโลหิตสูง (AOR 4.34, 95%CI: 1.37-13.71, p = .013); ภาวะช็อค (AOR 3.95, 95%CI: 1.73-8.99, p = .001); และผู้ป่วยที่ได้รับยาปฏิชีวนะ (AOR 5.33, 95%CI: 2.32-12.26, p<.001) การจากศึกษาทราบถึงอุบัติการณ์ของภาวะไตวายเฉียบพลันจากสารทึบรังสีและปัจจัยที่สัมพันธ์ซึ่งสามารถใช้เป็นแนวทางในการคัดกรองและดูแลผู้ป่วยให้ปลอดภัยต่อไป

คำสำคัญ : ภาวะไตวายเฉียบพลันจากสารทึบรังสี, กล้ามเนื้อหัวใจขาดเลือด, การขยายหลอดเลือดหัวใจ

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วันที่รับ (received) 15 มี.ค. 2567 วันที่แก้ไขเสร็จ (revised) 13 พ.ค. 2567 วันที่ตอบรับ (accepted) 25 พ.ค. 2567

Abstract

This study aimed to investigate the incidence and factors associated with Contrast-Induced Nephropathy (CIN) in patients with ST-segment Elevation Myocardial Infarction (STEMI) who underwent emergency Percutaneous Coronary Intervention (PCI) in the Thai population following the administration of contrast media (CM). The research was conducted retrospectively on 280 patients who experienced a creatinine level increase of more than 25% or exceeding 0.5 mg/dl. within 48-72 hours after receiving CM agents. Simple and multiple binary logistic regression analyses were employed to predict factors associated with CIN following contrast media exposure. Crude and Adjusted Odds Ratios (AOR) with 95% confidence intervals (CI) were utilized to demonstrate the associations between independent factors and CIN. The results revealed an incidence of CIN of 16.4%. Factors associated with CIN included CM volume exceeding 160 ml (AOR 4.66, 95%CI: 2.02-10.71, p<.001); heart failure (AOR 4.43, 95%CI: 1.80-10.93, p=.001); hypertension (AOR 4.34, 95%CI: 1.37-13.71, p=.013); anemia (AOR 3.95, 95%CI: 1.73-8.99, p=.001); and patients receiving antibiotic therapy (AOR 5.33, 95%CI: 2.32-12.26, p<.001). The study findings provide insights into the incidence of CIN and associated factors, serving as guidelines for screening and ensuring safe patient care in the future.

Keywords: contrast-induced nephropathy, myocardial infarction, coronary artery intervention.

Introduction

Contrast-induced nephropathy (CIN) is a frequent adverse effect of percutaneous coronary intervention (PCI) and can lead to renal function impairment and eventually hemodialysis if adequate preventive treatments are not provided. This adverse outcome from CIN can have negative impacts on one's quality of life, health expenses, morbidity, and increasing mortality rate.[1, 2] The incidence of CIN has been reported to be as high as 10-13% in patients undergoing PCI,[3,4] and it could even rise to 20-30% if significant risk factors are present.[5] This is particularly observed in patients with pre-existing chronic kidney disease (CKD) and congestive heart failure (CHF) before contrast media exposure.[3,6] Previous studies have found varying factors associated with CIN, depending on the studied population, these include patient-related factors such as advanced age, Diabetes Mellitus, Hypertension, Anemia, CHF, and CKD. Clinical setting-related factors include urgency or emergency setting, cardiogenic shock, and the use of nephrotoxic drugs. Procedure-related factors include the type and volume of CM used and the utilization of an Intra-Aortic Balloon Pump (IABP).[3,6-8]

Immediate revascularization aims to rapidly restore blood flow to the obstructed coronary artery, preserving viable heart tissue, thereby enhancing the STEMI patient's chances of survival and decreasing the likelihood of potential complications such as cardiogenic shock, acute heart failure, arrhythmia, and cardiac arrest,[9,10] and reduction of renal replacement therapy in STEMI patients.[11] This is due to the limited opportunity to implement preventive measures for CIN before the emergency procedure takes place and there is data suggesting that excessive hydration is associated with an increased risk of CIN and death in patients with pre-existing renal insufficiency undergoing PCI.[12,13] Currently, there are gap in knowledge regarding CIN within the Thai population pertains to the limited understanding of its incidence, risk factors, and optimal management strategies specifically tailored to this demographic. Closing this knowledge gap would

require further research studies and clinical investigations focused on CIN within the context of the Thai population, along with the development of evidence-based practices to improve patient outcomes.

Objectives

1. To study the Incidence of CIN after Emergency PCI in Thai Patients with STEMI.

2. To investigate the Associated factors with CIN after emergency PCI.

Research hypothesis

1. What is the true incidence of CIN after emergency PCI in the Thai population?

2. What factors are associated with CIN after emergency PCI?

Conceptual framework of the research

The research framework was developed from a literature review and the application of various theories such as Biomedical, Risk Stratification, and Pharmacological Theory. By integrating these theoretical perspectives, researchers can create a comprehensive conceptual framework for studying CIN in patients with STEMI after emergency PCI.

Methodology

A retrospective review study was conducted involving patients who had been admitted at cardiac care unit, Siriraj Hospital, Bangkok, Thailand, from June 2015 to August 2019. Population consists of patients with STEMI who underwent emergency PCI, aged 18 years and older, and met the inclusion criteria. The sample size was calculated through a literature review and preliminary statistics on CIN, estimated at approximately 20% from patient records. Due to the unknown population size and proportion, the formula $n = (\text{z-score})^2 \times \text{StdDev} \times (1-\text{StdDev})/(E)^2$ was utilized at a 95% confidence level. Z-score = 1.96, StdDev = 0.20, and E (margin of error) = 0.05 were used in the formula. This yielded a minimum sample size of 246 individuals. To account for a potential 10% loss of data, data were collected resulting in a total sample size of 280. STEMI is defined according to international guidelines regarding patients with persistent chest discomfort or other symptoms suggestive of ischemia and ST segment elevation in at least two contiguous leads or with the onset of a left bundle branch block on the electrocardiogram and a subsequent leave of biomarkers of myocardial necrosis.[14] CIN is defined as an increase in baseline serum creatinine by more than 25% or 0.5 mg/dl within 48-72 hours after CM exposure.1 Exclusion criteria included patients who missed the serum creatinine measurement before PCI within 72 hours; end-stage renal disease (ESRD) patients who had previously undergone dialysis; who received contrast media agents during the first 72 hours of PCI; and/or who needed emergency cardiac surgery after coronary angiography.

Research Tools and Instrument Quality

1. Demographics data, including age, gender, comorbidities, clinical presentation, echocardiography, and initial or baseline, as well as follow-up, laboratory test results.

2. Diagnosis of STEMI, including the affected area, cardiac catheterization results, amount of CM received, and the use of cardiac support devices.

3. Medications affecting kidney function or blood circulation, including ACEI, diuretics, antibiotics, inotropic or vasopressor, as well as data on hydration received to prevent CIN.

The research tools have been reviewed by 3 qualified experts in the relevant field, and content adjustments were made based on their feedback. Disease diagnoses and treatments, as well as health

conditions, were conducted and managed by physicians following standard medical practice guidelines.

Protection of Research Participants' Rights

This study protocol was approved by the Siriraj Institutional Review Board (SIRB), Faculty of Medicine, Siriraj Hospital, Mahidol University, under number SI 288/2019. Following ethical approval, the researchers obtained permission from the director of Siriraj Hospital to proceed with data collection from electronic medical records. They applied their eligibility criteria and collected data using a case record form. The data, anonymized using codes or other formats that could identify the research participants, were handled to maintain the confidentiality of the sample data. The findings will be reported in a summarized manner without identifying any individual patients.

Statistical analysis

The quantitative data, which were continuous with a normal distribution, were presented using mean ± SD and were compared using the independent t-test. The continuous variables with non-normal distributions were presented using a median and interquartile range and were compared using the Mann-Whitney U-test. The proportion of categorical variables data is presented as percentages and was compared using the Pearson Chi-square test. Simple and multiple binary logistic regression analyses were employed to investigate the factors associated with CIN. Crude and adjusted odds ratios (AOR) were used to determine the direction of associations between independent factors and CIN. The results are expressed as associated factors with a 95% confidence interval (CI). A p-value < 0.05 was considered statistically significant.

Results

A total of 280 participants who underwent emergency PCI were included for analysis. CIN developed in 46 patients (16.4%). Their average age was 64.0±12.6 years, and the majority of patients were male (73.7%). Baseline characteristics of the study population comparing between the group with CIN and the group without CIN as follows:

Table 1 Baseline characteristics, risk factors, laboratory results, clinical presentation, and procedure characteristic.

Characteristics	CIN (n = 46)	Non-CIN (n = 234)	p-value
Age, years (Mean±SD)	68.7±11.2	63.1±12.7	0.005
MAP, mmHg, Median(min,max)	68.5(60,113)	87.0(60,143)	0.001
Diabetes mellitus, n(%)	35(76.1)	107(45.7)	<0.001
Hypertension, n(%)	41(89.1)	143(61.1)	<0.001
Chronic kidney disease, n(%)	17(37.0)	35(15.0)	<0.001
Anemia, n(%)	23(50.0)	48(20.5)	<0.001
Prior myocardial infarction, n(%)	10(21.7)	20(8.5)	0.016
Heart failure, n(%)	37(80.4)	87(35.9)	<0.001
Cardiogenic shock, n(%)	30(65.2)	89(38.0)	0.001
Cardiac arrest, n(%)	14(30.4)	25(10.7)	<0.001

Table 1 Baseline characteristics, risk factors, laboratory results, clinical presentation, and procedure characteristic.

Characteristics		CIN (n = 46)	Non-CIN (n = 234)	p-value
LVEF, % (Mean±SD)		37.3±10.4	48.2±11.6	<0.001
Hb, mg/dl (Mean±SD)		12.6±2.5	13.7±2.3	0.016
Cr baseline, (mg/dl) Median(min,max)		1.24(0.63,3.37)	1.01(0.46,3.20)	<0.001
eGFR, ml/min/1.73 m2 (Mean±SD)		54.6±23.9	74.6±21.7	<0.001
Infraction part, n(%)	Anterior wall	31(67.4)	111(47.4)	0.013
	Lateral wall	9(16.6)	27(11.5)	0.137
	Posterior wall	2(4.3)	26(11.1)	0.162
	Inferior wall	13(28.3)	117(50.0)	0.007
	RV infarction	3(6.5)	36(15.4)	0.112
LVEDP (mmHg) Mean±SD		30.0±9.0	24.5±8.5	<0.001
Stent at, n(%)	LAD	32(69.6)	11(47.0)	0.005
	RCA	11(23.9)	107(45.7)	0.006
	LCx	6(13.0)	19(8.1)	0.268
Contrast volume, ml (Mean±SD)		186.6±34.9	147.0±35.9	<0.001
IABP, n(%)		20(43.5)	44(18.8)	<0.001

Abbreviations: CIN = contrast-induced nephropathy; BMI = Body Mass Index; MAP = mean arterial pressure; LVEF = left ventricular ejection fraction; Hb = hemoglobin; eGFR = estimated glomerular filtration rate; RV infarction = right ventricular infarction; LVEDP = left ventricular end diastolic pressure; LAD = left anterior descending artery; RCA = Right coronary artery; LCx = left circumflex artery; IABP = Intra-aortic balloon pump; *Significant difference with p-value < 0.05*

Table 1. The CIN group were older and had an underlying disease such as DM, HT, CKD stage 3 or above, or eGFR less than 60 ml/min/1.73 m^2, Anemia and prior myocardial infarction. The patients with CIN were more likely to have CHF, cardiogenic shock, and cardiac arrest. In addition, decreasing LVEF could significantly develop with CIN. As regards the laboratory investigation of CIN development, the mean± SD of Hb level was significantly lower than patients without CIN. Furthermore, the levels of serum creatinine were slightly higher in patients developing CIN than in those who did not. Most CIN patients had an anterior wall myocardial infarction which refers to the obstruction of the LAD. In contrast, the posterior wall, lateral wall, inferior and right ventricular infarction did not develop CIN significantly. The patients with CIN had higher LVEDP and were more likely to require an IABP.

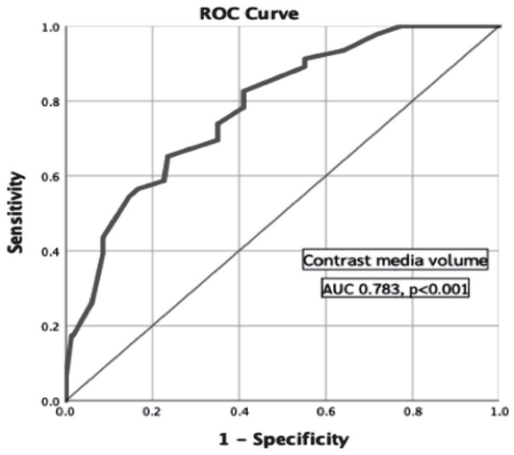

Figure 1. The contrast media agent received was used to generate the areas under the receiver operating characteristic curves (AUROC) to determine the optimal cut-off point in this study. Contrast agent received exceeding 160 milliliters is correlated with an increased risk of CIN (sensitivity 75.91, specificity 65.96, p<0.001)

Table 2 Medical treatment before receiving PCI and after PCI within 72 hours.

Medication		CIN (n = 46)	Non-CIN (n = 234)	p-value
Before PCI	ACEI, n(%)	7 (15.2)	91 (9.0)	0.307
	Loop diuretic, n(%)	13 (28.3)	23 (9.8)	0.002
	Inotropic/Vasopressor, n(%)	12 (26.1)	41 (17.5)	0.175
After PCI within 72 hours				
	ACEI, n(%)	6 (13.0)	109 (46.6)	<0.001
	Loop diuretic, n(%)	35 (76.1)	85 (36.3)	<0.001
	Ceftriaxone, n(%)	13 (28.3)	39 (16.7)	0.101
	Piperacillin/tazobactam, n(%)	15(32.6)	11(4.7)	<0.001
	Meropenem, n(%)	8 (17.4)	5 (2.1)	<0.001
	Vancomycin, n(%)	6 (13)	5(2.1)	0.003
	Inotropic or Vasopressor, n(%)	28 (60.9)	122 (52.1)	0.278
	NSS, ml (in 24 hours), Median (min, max)	490 (60,1300)	1100 (100,2760)	<0.001

Abbreviations: PCI = percutaneous coronary intervention; IV fluid = intravenous fluid; NSS = normal saline solution; ACEI = Angiotensin-converting enzyme inhibitors; Significant difference with p-value < 0.05

Table 2. CIN groups had higher proportion of being given loop diuretics both prior to and after PCI, and using antibiotics after PCI than non-CIN patients and Patients who received a lower volume of NSS had a higher chance of developing CIN. Nevertheless, the administration of inotropic and vasopressor showed no statistically significant association with the occurrence of CIN.

Table 3 Factors associated with CIN development in patients with STEMI undergoing emergency coronary angiography and/or percutaneous coronary intervention.

Factors	Crude OR (95%CI)	p-value	AOR (95%CI)	p-value
CM volume > 160ml	5.25(2.58-10.69)	<0.001	4.66(2.02-10.71)	<0.001
CHF	7.34(3.38-15.95)	<0.001	4.43(1.80-10.93)	0.001
Hypertension	5.22(1.99-13.69)	0.001	4.34(1.37-13.71)	0.013
Anemia	3.86(2.00-7.49)	<0.001	3.95(1.73-8.99)	0.001
Antibiotic usage	8.89(4.36-18.11)	<0.001	5.33(2.32-12.26)	<0.001

Abbreviations: OR = odds ratio; AOR = Adjusted odds ratio; 95%CI = 95%confidence interval; CM = contrast media; CHF = congestive heart failure; Significant difference with p-value < 0.05

Table3. Crude and adjusted odds ratio was used to determine the direction of associations between independent factors and CIN. Five factors were positively associated with CIN: receiving CM in volumes exceeding 160 milliliters (AOR 4.655, 95%CI:2.023-10.711, p< 0.001); CHF (AOR 4.433, 95%CI:1.798-10.927, p = 0.001); HT (AOR 4.335, 95%CI:1.370-13.711, p = 0.013); Anemia (AOR 3.946, 95%CI:1.731-8.994, p = 0.001); Antibiotic usage (AOR 5.332, 95%CI:2.318-12.264, p < 0.001); and were significantly associated with CIN development.

Discussion

STEMI patients who undergo emergency PCI have a higher incidence of CIN compared to patients undergoing other types of PCI procedures. Several significant factors associated with CIN were identified in this study, including receiving a CM volume greater than 160 ml, CHF, HT, anemia, and antibiotic usage. Additionally, CIN is indicative of a poorer quality of life for affected patients, as evidenced by longer hospital stays, an increased need for dialysis, and its broader impact on the healthcare system and mortality rates.

The incidence of CIN was 16.4% in this study. Numerous prior studies have reported CIN incidences in approximately 10-20% of patients following PCI.[3-5,7] The incidence could exceed 20% in elderly patients with pre-existing renal impairment,[15] particularly when combined with DM or CHF.5 In STEMI patients with both CHF and LVEF < 40% following PCI, a high incidence rate of 36.5% for CIN was observed.[16]

The exact pathophysiology of CIN remains unclear. CM directly affects the kidney tubular epithelial cells and vascular endothelial cells, leading to cytotoxic effects. CM triggers the development of vacuoles in kidney tubular cells through pinocytosis, causing osmotic nephrosis. This process is accompanied by mitochondrial dysfunction, resulting in the generation of reactive oxygen species (ROS) and apoptosis. The direct toxicity of CM results in the degradation of the tubular brush border, disruption of cell membrane integrity, and shedding of tubular epithelial cells into the lumen.[17,18] The harmful effects of contrast particles on endothelial cells result in elevated levels of endothelin and adenosine, along with reduced levels of nitric oxide (NO) and prostaglandins. As these changes persist, hypoxia and ischemia can develop. The dose of CM is important in the development of CIN, especially in individuals with pre-existing kidney dysfunction.[18] When the heart muscle lacks blood supply due to blockage in the coronary arteries, it leads to subsequent CHF. This results in a decrease in cardiac output due to reduced

contractility,[19] leading to hemodynamic instability and a reduced GFR.[20] CHF is a strong independent factor and an effective predictor of CIN[21]

Hypertension, as a chronic condition, which can ultimately result in kidney damage and cardiovascular disease. These mechanisms encompass factors such as salt and volume expansion, sympathetic nervous system hyperactivity, an upregulated renin-angiotensin-aldosterone system, vascular remodeling, endothelial dysfunction, and various signaling molecules. They can induce various alterations in the vasculature and kidney function, leading to structural changes in the small blood vessels of the kidneys, causing them to narrow and stiffen. This narrowing reduces blood flow into the kidneys, reduced renal perfusion can make the kidneys more vulnerable to injury when exposed to CM.[22]

In individuals with anemia, there is a reduction in red blood cells, resulting in a decreased oxygen-carrying capacity of the blood. This leads to insufficient oxygen delivery to cause renal medullary hypoxia. Simultaneously, exposure to CM causes blood vessels to constrict, reducing blood circulation in the kidneys. Consequently, anemia may make the kidneys more susceptible to injury when they come into contact with CM, which carries a risk of renal impairment.[23] Sreenivasan et al. conducted a study suggesting that the severity of anemia impacts the development of CIN24, and anemia significantly increases the risk of developing CIN in patients undergoing CAG or PCI, particularly in patients with CKD, as found in Wei Liang et al.'s study.[25]

In patients undergoing complex PCI with a prolonged procedure duration, there is an increased risk of bacterial contamination, particularly when the procedure involves the femoral artery approach.[26] In patients suspected of sepsis, especially in cases of hemodynamic instability, it is recommended to empirically administer at least one type of broad-spectrum antimicrobial following international guidelines when the causative pathogen is unknown.[27] It is well-established that antimicrobials can be nephrotoxic and can further increase the risk, especially in patients receiving CM during PCI.[30] Vancomycin is commonly used to treat infections caused by staphylococcal and streptococcal bacteria. However, acute tubular necrosis and acute interstitial nephritis are mechanisms that can lead to vancomycin-related acute kidney injury (AKI). The combination of vancomycin and piperacillin-tazobactam was associated with a higher incidence of AKI when compared to using vancomycin alone or in combination with meropenem in several studies.[28]

Limitations

First, this observational retrospective study is conducted at a single center and acknowledges the limitation of a relatively small study population, which involves the usual bias of such a retrospective analysis. However, it's worth noting that our patients are reflective of real-world emergency PCI cases. Second, CIN's incidence and influencing factors can differ based on various clinical practice guidelines and situations. Third, the study population focused specifically on patients who underwent emergency PCI.

Summary and Explanation

STEMI patients who undergo emergency PCI have a higher incidence of CIN. Several significant factors associated with CIN were identified in this study, including receiving a contrast medium volume greater than 160 ml, CHF, HT, anemia, and antibiotic usage. Additionally, CIN is indicative of a poorer quality of life for affected patients, as evidenced by longer hospital stays, an increased need for dialysis, and its broader impact on the healthcare system and

mortality rates.

Suggestions obtained from research: The research found a high incidence of CIN following emergency PCI. Based on these findings, we recommend implementing early screening for high-risk patients, particularly those with HT, CHF, and anemia. It is important to limit the volume of CM used during PCI procedures and to be cautious when prescribing nephrotoxic drugs, such as certain antibiotics. However, other factors may also play a role. It is recommended to regularly review and update clinical practice guidelines to reduce the incidence of contrast-induced nephropathy in patients undergoing emergency PCI, thereby improving overall patient outcomes.

Suggestions for further Research: Conduct studies on larger and multi-center, diverse populations to validate current findings and refine risk stratification models. Additionally, investigate the occurrence of CIN and compare it with other cardiovascular procedures, such as Transcatheter Aortic Valve Implantation.

Conflict of interest

The authors declared that we have no conflicts of interest to disclose.

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