PREVALENCE OF CONTRAST-INDUCED ACUTE KIDNEY INJURY AFTER PRIMARY PERCUTANEOUS CORONARY INTERVENTION: A SYSTEMATIC REVIEW

Muhammad Atiq Ur Rehman Khan[1], Muhammad Adnan Khan[2], Muhamad Sajjad Sadiq[3], Adil Shabbir[4], Feroza Zarin[5]

1. Registrar cardiology Sligo Univ Hospital, Ireland
2. Registrar Endocrinology Mayo General Hospital Castlebar, Ireland
3. Consultant medicine Mayo General Hospital Castlebar, Ireland
4. Registrar endocrinology, Mayo General Hospital Castlebar Ireland
5. Sho Medicine Mayo General Hospital Castlebar, Ireland

ARTICLE INFO

Key words:
Prevalence, Acute Kidney Injury due to contrast, Primary PCI, Cardiovascular Disease, Systematic Review

Corresponding Author:
Muhamad Sajjad Sadiq,
Consultant medicine Mayo General Hospital Castlebar, Ireland
Email: fatimazubair9@gmail.com

Vol 01 Issue 03
JULY-SEP 2023

ISSN Online: 2960-2599
ISSN Print: 2960-2580

SYSTEMATIC REVIEW

ABSTRACT:

Background: Contrast-induced acute kidney injury (CIN) is a frequent side effect of percutaneous coronary intervention (PCI) that is linked to increased rates of death and morbidity as well as extended hospital stays in patients with cardiovascular disease. **Objective:** To systematically review the current status of CIN in patients undergoing PCI. **Methods:** This Systematic review was done on 7 studies. A thorough investigation was conducted on contrast-induced nephropathy (CIN) or contrast-induced acute kidney damage (CI-AKI) in patients receiving PCI and/or coronary angiography; the results are freely available on PubMed, Google Scholar, MEDLINE, and the internet. All articles which were published in English using Boolean Operators (AND, OR, NOT or AND NOT), were searched with the help of keywords like CIN, CI, AKI, PCI, etc. All free full-length articles that had distinct goals, were full-length, and were published in the English language in a few search engines were selected, and systematic review and review articles and all other articles with ambiguous results were excluded. **Results: In the** current study, it was found that the prevalence of CI-AKI /CIN in patients who underwent PCI is reported as 2.1%–24.8%, depending upon the severity, type of PCI, and other factors. **Conclusion**: It is concluded that CIN is high in many of the studies, so it can be minimized using the minimum volume of the contrast media, particularly in high-risk patients. So, by controlling causative factors such as anemia, age, creatinine, etc., the CIN can be reduced..

INTRODUCTION

Myocardial Infarction (MI) is a common risk factor of Heart Failure (HF) globally. HF has been known for about 50 years to be a factor in a poor prognosis after myocardial infarction (MI), leading to ineffective cardiac repair after therapeutic interventions.[1] The appropriate mode of management for ischemic coronary artery disease (CAD) is controversial in literature. Several Randomized Controlled Trials (RCTs) and meta-analyses have concluded that the optimum treatment approach depends largely on severity of disease and complications involved. [2] Percutaneous coronary intervention (PCI) is the most widely utilized invasive technique for treating patients with coronary heart disease, and it has a strong body of evidence supporting it.[3] Unfavorable procedural results might happen during the PCI. [4] In patients having coronary angiography and/or PCI, a rise in the use of iodinated contrast media, such as iodixanol, iohexol, iopromide, and iopamidol, may

infrequently result in contrast-induced nephropathy (CIN) or contrast-induced acute kidney damage (CI-AKI).[5]

The CIN is a frequent side effect of PCI that is linked to increased rates of death and morbidity as well as extended hospital stays in patients with ST elevation myocardial infarction (STEMI).[6] When contrast media is administered parenterally and there are no other apparent reasons, the CI-AKI or CIN is characterized by a dramatic decline in renal function within a few days.[7] A spike in serum creatinine of at least 0.5 mg/dL or a 25% relative rise within 48 hours after contrast exposure is referred to as CI-AKI or CIN.[8] Patients who undergone percutaneous coronary intervention (PCI) are expected to have a prevalence of 12% for CI-AKI being the most prevalent kind of iatrogenic kidney impairment.[7] While often self-resolving, CI-AKI is linked to higher short- and long-term morbidity and mortality in individuals.[7] However, being aware of potential issues and promptly managing them may save a patient's life and is crucial for practising cardiologists.[4] Hence, this systematic review was planned to see the current status of CI-AKI/ CIN in patients undergoing PCI, so that it can be managed through proper prophylactic strategies.

Materials and Methods

Study design: Systematic review

Reporting Method: A thorough investigation was conducted on contrast-induced nephropathy (CIN) or contrast-induced acute kidney damage (CI-AKI) in patients receiving PCI and/or coronary angiography; the results are freely available on PubMed, Google Scholar, MEDLINE, and the internet. The article's inclusion and eligibility were reported using the usual procedures as per the Preferred Reporting Items for Systematic Review and Meta-Analysis Statement (PRISMA).

Search Strategy and Selection Criteria

All articles which were published in English language using Boolean Operators (AND, OR, NOT or AND NOT), the selected engines were searched with the help of keywords like, CIN, CI, AKI, PCI etc.

Selection criteria

All free full-length articles had distinct goals, were full-length, and were published in the English language in a few search engines were selected and systematic review and review articles and all other articles with ambiguous results were excluded.

Flow chart of studies

We followed PRISMA flow chart.

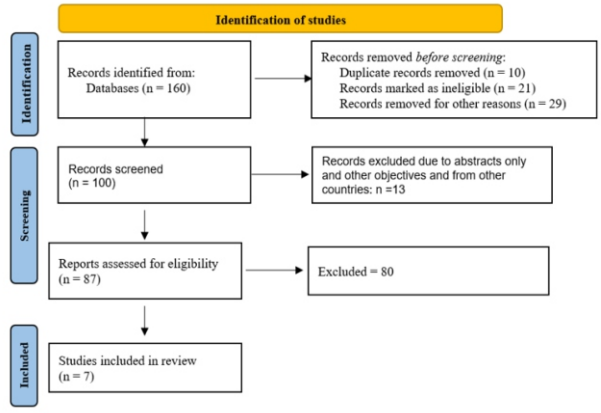

Fig-1: PRISMA criteria

Data Extraction Technique:

In order to gather information from the research that made the short list, a data extraction form was created. The table had the following details: population, prevalence of CIN, sample size, year, study strategy, and author information. A comprehensive conversation was conducted to address any differences in the supplied data and arrange for the papers to be evaluated again. The findings were shown as a table.

RESULTS

A total of 7 studies were taken in this review, where total sample size became 5470 and prevalence of CI-AKI or CIN was 834(15.25%). Among these 7 studies, 5 studies reported prospective trial, one was observational study and 1 study did not report any study design. A study from Pakistan was done on 237 cases of acute coronary syndrome (ACS) at Aga Khan Hospital, all patients underwent percutaneous coronary intervention (PCI). The mean pre-PCI creatinine was 0.88±0.20 mg/dl, according to their study. Non-ionic contrast was used in 235 individuals, or 99.2% of the total. The contrast's average volume was 201.18±79.15 millilitres. The mean post PCI (48 hours) creatinine was 0.95±0.27 mg/dl. A total of 5(2.1%) developed contrast-induced nephropathy.[9] **Table -1**

In a study from United States of America (USA), a total of 2968 patients of STEMI having PCI, got CI-AKI as 16.1%.[8] It was also observed in a study from Jammu & Kashmir that 12.6% had CI-AKI who underwent percutaneous coronary intervention.[10] An Indian study reported 10.66% frequency of CI-AKI in cases underwent PCI for CAD. Multivariate analysis revealed that the usage of ace inhibitors, lower ejection fraction, number of stents, diabetes mellitus, and contrast dosage (odds ratio 1.048, 95% CI) were all substantially linked to CIN.[11] One more study was done in STEMI who underwent PCI had CIN as 13.5%.[6] One of the highest CIN was reported by 2 studies , one from China (24.8%)[12] and Brazil (23.8%)[13], done on 308 cases of High risk STEMI or NSTE-ACS and 201 STEMI cases respectively. **Table -1**

Table -1: Prevalence of CI-AKI / CIN in patients underwent PCI

Study	Country / region	N	n(%)	Study design	Population	REF
(Babar et al., 2022)	Pakistan	237	5(2.1%)	Prospective	ACS	9
(Narula et al., 2014)	USA	2968	479 (16.1%)	Prospective	STEMI	8
(Peer et al., 2016)	Jammu & Kashmir	222	28 (12.6%)	Observational		10
(Singhal et al., 2017)	India	300	32 (10.66%)	Prospective	CAD	11
(Rencuzogu-llari et al., 2018)	Turkey	1234	166 (13.5%)		STEMI	6
(Liu et al., 2015)	China	308	76(24.8%)	Prospective	STEMI / NSTE-ACS	12
(Santos et al., 2015)	Brazil	201	48 (23.8%)	Prospective	STEMI patients	13
Total		**5470**	**843 (15.25%)**			

DISCUSSION

Cardiovascular disease (CVD) is the main cause of death and morbidity globally and a global health concern that is spreading pandemic proportions in both industrialised and developing nations.[14, 15] The incidence of CVD is among the highest in the world among the nations of South Asia.[16] Over the past several decades, a number of high-income nations have seen a decline in the rates of cardiovascular disease-related fatalities; nevertheless, low- and middle-income countries have seen a rise in these deaths, accounting for around 80% of the burden.[17] Despite the significance of cardiovascular diseases in low- and middle-income countries, South Asia, particularly Pakistan, pays little attention to the prevention of cardiovascular disease risk factors.[17] According to estimates, more people with atherosclerotic cardiovascular disease (CVD) will live in this area of the globe by 2020 than in any other.[18] A major consequence of the widespread use of PCI technology in patients with coronary artery disease (CAD) is contrast-induced acute kidney damage (CI-AKI).[19] The creatinine level usually peaks three to five days after the surgery and returns to baseline or nearly baseline value in one to three weeks. It happens within 24 to 48 hours of the exposure.[20] In current study it found that the prevalence of CI-AKI / CIN in patients underwent PCI is reported as 2.1%[9] -

24.8%[12], depending upon the severity, type of PCI and other factors. According to a research, the use of minimum contrast media volume, PCI may lower the occurrence of CIN, especially in patients who are at high risk.[21]

According to our present knowledge of the illness, there are several significant questions about the pathophysiology and preventative measures, as well as a complex aetiology.[22] The kind and amount of the contrast agent, together with patient-related variables such advanced age, anaemia, sex, diabetes mellitus, congestive heart failure, CKD, and decreased effective circulation volume, are risk factors for development.[23, 24] Another research found that the following factors were associated with a higher risk of CI-AKI: creatinine clearance <60 mL/min, age, age-related left anterior descending infarct related artery, anaemia, white blood cell count, and history of congestive heart failure.[8] Hence, by further studies, and controlling these factors CIN can be reduced.

Conclusion:

It is concluded that CIN is high in many of the studies, so it can be minimized using the minimum volume of the contrast media, particularly in high-risk patients. So, by controlling causative factors such as anemia, age, creatinine, etc., the CIN can be reduced.

AUTHORS CONTRIBUTION

MAURK: Idea conception, write up, **MAK:** Literature search / writing, **MSS:** Literature search, **AS:** Write up, **FZ:** Literature and Reference

REFERENCES

1. Cahill TJ, Kharbanda RK. Heart failure after myocardial infarction in the era of primary percutaneous coronary intervention: Mechanisms, incidence and identification of patients at risk. World J Cardiol. 2017;9 (5):407-15.

2. Spadaccio C, Benedetto U. Coronary artery bypass grafting (CABG) vs. percutaneous coronary intervention (PCI) in the treatment of multivessel coronary disease: quo vadis? - a review of the evidences on coronary artery disease. Ann Cardiothorac Surg. 2018;7 (4):506-15.

3. Banning AP, Baumbach A, Blackman D, Curzen N, Devadathan S, Fraser D, et al. Percutaneous coronary intervention in the UK: recommendations for good practice 2015. Heart. 2015;101(Suppl 3):1-13.

4. Giannini F, Candilio L, Mitomo S, Ruparelia N, Chieffo A, Baldetti L, et al. A practical approach to the management of complications during percutaneous coronary intervention. JACC: Cardiovascular Interventions. 2018;11(18):1797-810.

5. Xu J, Zhang M, Ni Y, Shi J, Gao R, Wang F, et al. Impact of low hemoglobin on the development of contrast-induced nephropathy: A retrospective cohort study. Exp Ther Med. 2016;12(2):603-10.

6. Rencuzogullari I, Çağdaş M, Karakoyun S, Karabağ Y, Yesin M, Gürsoy MO, et al. Association of Syntax Score II with contrast-induced nephropathy and hemodialysis requirement in patients with ST segment elevation myocardial infarction undergoing primary percutaneous coronary intervention. Korean Circulation J. 2018;48(1):59-70.

7. Faggioni M, Mehran R. Preventing contrast-induced renal failure: a guide. Interventional Cardiology Review. 2016;11(2):98.

8. Narula A, Mehran R, Weisz G, Dangas GD, Yu J,

Genereux P, et al. Contrast-induced acute kidney injury after primary percutaneous coronary intervention: results from the HORIZONS-AMI substudy. Europ heart J. 2014;35(23):1533-40.

9. Babar SM, Kazmi KA, Tai JM, Memon FF, Uzair S, Bukhari S, et al. Contrast Induced Nephropathy in Patients with Acute Coronary Syndrome Undergoing Per-cutaneous Coronary Intervention. Pak J Med Health Sci. 2022;16(05):1362-.

10. Peer S, Choh NA, Gojwari TA. Incidence of contrast-induced nephropathy a prospective study. Journal of Renal Injury Prevention. 2016;6(3):192-8.

11. Singhal G, Pathak V, Kumar M. Incidence of contrast induced acute kidney injury in patients undergoing percutaneous coronary intervention in North Indian population. J Indian Coll Cardiol. 2017;7(4):143-8.

12. Liu Y, Lin L, Li Y, Li H, Wu D-X, Zhao J-b, et al. Relationship between the urine flow rate and risk of contrast-induced nephropathy after emergent percutaneous coronary intervention. Medicine. 2015;94(50).

13. Santos PR, Carneiro Neto JD, Arcanjo FPN, Carneiro JKR, Carneiro RCdCP, Amaral CLd. Contrast-induced nephropathy after primary angioplasty for acute myocardial infarction. Brazilian J Nephrol. 2015;37:439-45.

14. Murray CJ, Lopez AD. Measuring the global burden of disease. N Engl J Med. 2013;369(5):448-57.

15. Islam SM, Purnat TD, Phuong NT, Mwingira U, Schacht K, Fröschl G. Non-communicable diseases (NCDs) in developing countries: a symposium report. Global Health. 2014;10:81-90.

16. Kumar S. Cardiovascular disease and its determinants: Public health issue. J Clin Med Ther. 2017;2(1):1-5.

17. Barolia R, Sayani AH. Risk factors of cardiovascular disease and its recommendations in Pakistani context. J Pak Med Assoc. 2017;67(11):1723-9.

18. Karim MA, Majumder AAS, Islam KQ, Alam MB, Paul ML, Islam MS, et al. Risk factors and in-hospital outcome of acute ST segment elevation myocardial infarction in young Bangladeshi adults. BMC Cardiovascular Disorders. 2015;15(1):1-8.

19. Yuan Y, Qiu H, Hu X-Y, Luo T, Gao X-J, Zhao X-Y, et al. Risk Factors of Contrast-induced Acute Kidney Injury in Patients Undergoing Emergency Percutaneous Coronary Intervention. Chinese Med J. 2017;130(1):45-50.

20. Monwarul A. Impact of Low Hemoglobin on Contrast-Induced Nephropathy After Percutaneous Coronary Intervention. (Cardiovasc J. 2012;5(1):30-6.

21. Ebisawa S, Kurita T, Tanaka N, Nasu K, Kimura M, Ito T, et al. Impact of minimum contrast media volumes during elective percutaneous coronary intervention for prevention of contrast-induced nephropathy in patients with stable coronary artery disease. Cardiovascular intervention and therapeutics. 2016;31:13-20.

22. Căldăraru CD, Dobreanu D, Dogaru M, Olariu O, Dogaru G. Risk factors for contrast-induced

nephropathy after coronary angiography. Farmacia. 2014;4:711-20.

23. Ohno Y, Maekawa Y, Miyata H, Inoue S, Ishikawa S, Sueyoshi K, et al. Impact of periprocedural bleeding on incidence of contrast-induced acute kidney injury in patients treated with percutaneous coronary intervention. J Am Coll Cardiol. 2013;62(14):1260-6.

24. Medalion B, Cohen H, Assali A, Vaknin Assa H, Farkash A, Snir E, et al. The effect of cardiac angiography timing, contrast media dose, and preoperative renal function on acute renal failure after coronary artery bypass grafting. J Thorac Cardiovasc Surg. 2010;139(6):1539-44.