IJC Heart & Vasculature 21 (2018) 78–79

Contents lists available at ScienceDirect

IJC Heart & Vasculature

journal homepage: http://www.journals.elsevier.com/ijc-heart-and-vasculature

Correspondence

An intriguing relation between atrial fibrillation and contrast-induced nephropathy

Kumar Ashish [a], Dhrubajyoti Bandyopadhyay [b,*], Raktim K. Ghosh [c], Jian Liang Tan [a], Subhasish Bose [d]

[a] Crozer-Chester Medical Center, Upland, USA
[b] Mount Sinai St Luke's Roosevelt Hospital, New York, USA
[c] MetroHealth, Case Western Reserve University, Cleveland, OH, USA
[d] University of Virginia, USA

ARTICLE INFO

Article history:
Received 13 October 2018
Accepted 16 October 2018
Available online 24 October 2018

Keywords:
Atrial fibrillation
Contrast induced nephropathy

Atrial fibrillation (A-Fib) is the most common sustained cardiac arrhythmia leading to increased morbidity and mortality. It is a significant health problem that around 5.0 million Americans experience in their lifetime. Its prevalence increases with age and affects Caucasians more compared to African-Americans [1].

Acute kidney injury (AKI) is an important renal issue that may have various causes including contrast-induced nephropathy (CIN). Due to the increase in interventional cardiac procedures and radiological investigation with intravenous iodine-based contrast media (CM), CIN has turned out to be the third most common cause for hospital-acquired AKI and represents 12% of cases. CIN can cause various debilitating in-hospital complications including an overall mortality rate of ~20% and may tend to have long-term loss of kidney functions, in addition to higher financial cost. Even after modifying the co-morbidities, CIN leads to five-fold higher in-patient mortality than a patient who didn't develop CIN after receiving CM [2].

CM could be classified by their distinct osmolality: high-osmolar (~1000–2500 mosmol/kg H2O), low-osmolar (~400–800 mosmol/kg H2O) and iso-osmolar (~290–320 mosmol/kg H2O) CM. Radio-opacity of contrast agent relies on the amount of iodine on benzene derivatives. Both high osmolality and high viscosity have been implicated in putative hemodynamic effects that can lead to CIN. Studies have demonstrated that using low or iso-osmolar contrast agents or carbon dioxide is associated with less or no occurrences of CIN [2,3].

By definition, CIN is the elevation of serum creatinine (Scr) by ≥25% or >0.5 mg/dl from baseline within 48 h after excluding other possible risk factors for AKI such as nephrotoxins, hypotension, urinary obstruction, or atheromatous emboli. In most instances, it follows a self-limiting course with Scr levels maximum at day 3–5, and gradually AKI resolves within 7–10 days. There are several risk factors for CIN which are classified into two categories: patient and procedure-related. Patient-related risk factors are chronic kidney disease (CKD) [estimated glomerular filtration rate (eGFR) ≤ 60 ml/min], diabetes mellitus, age > 75 years, hypertension, hypotensive episode requiring inotropes, congestive heart failure (CHF), use of intra-aortic balloon pump, liver cirrhosis, and hypoalbuminemia. On the other hand, procedure-related risk factors comprise of high CM volume, high osmolality CM, ionic contrast, contrast viscosity, repeated use within 72 h and concomitant use of nephrotoxic drugs along with CM [4].

Contrast agents have interactions with various medications so the physicians should look for those interactions to mitigate the hazardous implication of CM. We may need to withhold few drugs before contrast based procedures like metformin, renin inhibitors, angiotensin-converting enzyme inhibitors (ACEI), angiotensin receptor blocker (ARBs), non-steroidal anti-inflammatory drugs, loop diuretics and thiazide-like diuretics [5]. Primary angioplasty intervention in myocardial infarction cases have a higher risk of developing CIN compared to elective procedures. To prevent these implications, preemptive use of normal saline infusion and *N*-acetylcysteine may be helpful. Notably, it should be given many hours before starting the procedure [6].

Renal dysfunction is indisputably related to cardiovascular disease causing significant death and disability, typically when there is underlying acute coronary syndrome (ACS) [7]. Retrospective cohort study performed by Roubín et al. to contemplate possible reciprocal relation between a-fib and CIN, involving 1520 participants supported that CIN is an independent predictor of the occurrence of new-onset A-Fib (p-value < 0.001). Several hypotheses were suggested to explain this unidirectional association between CIN and A-Fib like influence on renin-angiotensin-aldosterone system and the inflammatory pathway [8]. Thus, CIN carries a potential risk for new-onset A-Fib. Therefore, CIN after CM exposure should make the physician more vigilant for the possible hazard of A-Fib in these subjects.

A-Fib prognosis is intertwined with underlying cardiovascular morbidity, and mortality and that, in turn, is significantly related to

* Corresponding author.
E-mail address: dhrubajyoti.bandyopadhyay@mountsinai.org (D. Bandyopadhyay).

https://doi.org/10.1016/j.ijcha.2018.10.005

CKD. Yanagisawa and colleagues conducted a retrospective study involving 110 participants who underwent cryoballoon-catheter ablation for A-Fib cure. They sought a relation between prognosis after the cryoballoon ablation of A-Fib and renal function. The study population was divided into three subgroups on the basis of their eGFR and CKD stage. After multivariate regression analysis, this study demonstrated the difference in outcomes following cryoballoon ablation in subjects by CKD stage. Low eGFR at baseline is a remarkable predictor of recurrence of paroxysmal A-Fib in patients who underwent cryoballoon ablation. A-Fib recurrence percentages of 7%, 12%, and 46% were found in stage 1, stage 2 and stage 3 CKD respectively. Nonetheless, the presence of non-pulmonary vein ectopic beats was exponentially enhanced in participants with renal dysfunction which could lead to unfavorable outcomes [9].

Another retrospective study was done by Balli et al., enrolled 650 consecutive ST-elevation myocardial infraction (STEMI) patients who underwent primary percutaneous coronary interventions to evaluate the potential relationship between A-Fib and development of CIN in these patients. Analysis of this study illustrated that A-Fib [95% confidence interval (CI): 2.789–17.293; $p < 0.001$], eGFR [95% CI: 0.957–0.989; $p = 0.001$] and left ventricular ejection fraction (LVEF) [95% CI: 0.935–0.991; $p = 0.010$] are independent predictors of CIN consequences in STEMI patients after cardiac catheterization procedures [10].

The pathogenesis and risk factors for CIN are multifactorial and remain poorly understood. Nevertheless, the estimation of an individual's risk profile is extremely important in the prevention of CIN occurrence, even if the patient's baseline renal dysfunction is minimal. Considering the above studies, we can assume that there is an important bilateral relationship between A-Fib and CIN. However, the question remains whether CIN increases the risk of A-Fib in patients without cardiovascular disease or vice-versa. The big shortcomings of previous studies were small sample size and retrospective nature of study; therefore, we need a larger sample size and additional prospective studies for better understanding of this issue.

Conflict of interest

There is no conflict of interest.

References

[1] S.S. Chugh, J.L. Blackshear, W.K. Shen, S.C. Hammill, B.J. Gersh, Epidemiology and natural history of atrial fibrillation: clinical implications, J. Am. Coll. Cardiol. 37 (2) (Feb 1, 2001) 371–378 (PMID: 11216949).

[2] E. Seeliger, M. Sendeski, C.S. Rihal, P.B. Persson, Contrast-induced kidney injury: mechanisms, risk factors, and prevention, Eur. Heart J. 33 (16) (Jan 19, 2012) 2007–2015 (PMID: 22267241).

[3] F. Ali, M.A. Mangi, H. Rehman, E. Kaluski, Use of carbon dioxide as an intravascular contrast agent: a review of current literature, World J. Cardiol. 9 (9) (Sep 26, 2017) 715 (PMID: 29081903).

[4] N.M. Mohammed, A. Mahfouz, K. Achkar, I.M. Rafie, R. Hajar, Contrast-induced nephropathy, Heart Views 14 (3) (Jul 2013) 106 (PMID: 24696755).

[5] S. Hiremath, J.F. Kayibanda, B.J. Chow, D. Fergusson, G.A. Knoll, W. Shabana, B. Lahey, O. McBride, A. Davis, A. Akbari, Drug discontinuation before contrast procedures and the effect on acute kidney injury and other clinical outcomes: a systematic review protocol, Syst. Rev. 7 (1) (Dec 2018) 34 (PMID: 29467030).

[6] G. Marenzi, E. Assanelli, I. Marana, G. Lauri, J. Campodonico, M. Grazi, M. De Metrio, S. Galli, F. Fabbiocchi, P. Montorsi, F. Veglia, N-acetylcysteine and contrast-induced nephropathy in primary angioplasty, N. Engl. J. Med. 354 (26) (Jun 29, 2006) 2773–2782 (PMID: 16807414).

[7] M.J. Sarnak, A.S. Levey, A.C. Schoolwerth, L. LeeHamm, P.A. McCullough, B.L. Kasiske, M.J. Klag, D.J. Spinosa, P.W. Wilson, Kidney disease as a risk factor for development of cardiovascular disease, Hypertension 42 (5) (2003 Nov) 1050–1065 (PMID: 14604997).

[8] S.R. Roubín, R.A. Abellas-Sequeiros, E.A. Assi, R.R. Yousef-Abumuaileq, M.R. Mañero, D.I. Álvarez, V. González-Salvado, R.G. Ferreiro, A.R. Diéguez, R.O. Sánchez, A.V. Lamela, Relation of contrast induced nephropathy to new onset atrial fibrillation in acute coronary syndrome, Am. J. Cardiol. 115 (5) (Mar 1, 2015) 587–591 (PMID: 25591897).

[9] S. Yanagisawa, Y. Inden, H. Kato, A. Fujii, Y. Mizutani, T. Ito, Y. Kamikubo, Y. Kanzaki, M. Ando, M. Hirai, R. Shibata, Impaired renal function is associated with recurrence after cryoballoon catheter ablation for paroxysmal atrial fibrillation: a potential effect of non-pulmonary vein foci, J. Cardiol. 69 (1) (Jan 1, 2017) 3–10 (PMID: 27499270).

[10] M. Ballı, H. Taşolar, M. Çetin, E. Hatem, Ç.E. Çağlıyan, T. Şeker, M. Çaylı, Is atrial fibrillation a risk factor for contrast-induced nephropathy in patients with ST-elevation myocardial infarction? J. Cardiol. 67 (4) (Apr 1, 2016) 327–330 (PMID: 26589269).