Original Article

Risk Factors of Contrast-induced Acute Kidney Injury in Patients Undergoing Emergency Percutaneous Coronary Intervention

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Abstract

Background: Previous studies of contrast-induced acute kidney injury (CI-AKI) were mostly based on selective percutaneous coronary intervention (PCI) cases, and risk factors of CI-AKI after emergency PCI are unclear. The aim of this study was to explore the risk factors of CI-AKI in a Chinese population undergoing emergency PCI.

Methods: A total of 1061 consecutive patients undergoing emergency PCI during January 2013 and June 2015 were enrolled and divided into CI-AKI and non-CI-AKI group. Univariable and multivariable analyses were used to identify the risk factors of CI-AKI in emergency PCI patients. CI-AKI was defined as an increase in serum creatinine ≥25% or ≥0.5 mg/dl (44.2 μmol/L) above baseline within 3 days after exposure to contrast medium.

Results: The incidence of CI-AKI in patients undergoing emergency PCI was 22.7% (241/1061). Logistic multivariable analysis showed that body surface area (BSA) (odds ratio [*OR*] 0.213, 95% confidence interval [*CI*]: 0.075–0.607, $P = 0.004$), history of myocardial infarction (MI) (*OR* 1.642, 95% *CI:* 1.079–2.499, $P = 0.021$), left ventricular ejection fraction (LVEF) (*OR* 0.969, 95% *CI:* 0.944–0.994, $P = 0.015$), hemoglobin (Hb) (*OR* 0.988, 95% *CI:* 0.976–1.000, $P = 0.045$), estimated glomerular filtration rate (*OR* 1.027, 95% *CI*: 1.018–1.037, $P < 0.001$), left anterior descending (LAD) stented (*OR* 1.464, 95% *CI*: 1.000–2.145, $P = 0.050$), aspirin (*OR* 0.097, 95%*CI*: 0.009–0.987, $P = 0.049$), and diuretics use (*OR* 1.850, 95% *CI*: 1.233–2.777, $P = 0.003$) were independent predictors of CI-AKI in patients undergoing emergency PCI.

Conclusion: History of MI, low BSA, LVEF and Hb level, LAD stented, and diuretics use are associated with increased risk of CI-AKI in patients undergoing emergency PCI.

Key words: Contrast-induced Acute Kidney Injury; Emergency Percutaneous Coronary Intervention; Risk Factors

Introduction

With wide application of percutaneous coronary intervention (PCI) technology in patients with coronary artery disease (CAD), contrast-induced acute kidney injury (CI-AKI) has become a serious complication and is the third leading cause of AKI in hospitalized patients.[1] In the past several years, the reported incidences of CI-AKI ranged from 2% to 30% due to different populations and CI-AKI definitions.[2,3] CI-AKI is associated with increased morbidity and mortality.[4-9] Watabe *et al*.'s study showed that CI-AKI was a significant incremental predictor of cardiovascular events at each stage of chronic kidney disease (CKD) in acute coronary syndrome (ACS) patients.[10] Emergency procedure was reported to be an independent risk factor of CI-AKI, and CI-AKI rate in emergency PCI patients is significantly higher than in those undergoing selective intervention.[11,12] However, previous studies of CI-AKI were mostly based on data from selective

Access this article online

Quick Response Code:

Website: www.cmj.org

DOI: 10.4103/0366-6999.196578

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Received: 06-09-2016 **Edited by:** Yi Cui
How to cite this article: Yuan Y, Qiu H, Hu XY, Luo T, Gao XJ, Zhao XY, Zhang J, Wu Y, Yan HB, Qiao SB, Yang YJ, Gao RL. Risk Factors of Contrast-induced Acute Kidney Injury in Patients Undergoing Emergency Percutaneous Coronary Intervention. Chin Med J 2017;130:45-50.

cases. To those undergoing emergency PCI, the estimation of the risk of CI-AKI is always limited for being pressed for time after admission, and so far, the risk factor profile of CI-AKI in emergency PCI patients is still unclear and needed to investigate. The aim of this study was to explore the risk factors of CI-AKI in a Chinese population undergoing emergency PCI.

Methods

Study population

From January 1, 2013, to June 30, 2015, patients who underwent emergency PCI at a single center (Fuwai Hospital, Beijing, China) were enrolled consecutively. The inclusion criterion was patients undergoing emergency PCI; the exclusion criteria were those (1) who contact with contrast medium <1 week before procedure, (2) allergic to iodinated contrast medium, (3) with severe heart failure(New York classification IV or cardiac stroke), serious valvular heart disease, or hemodynamic instability, (4) contact with nephrotoxic medicine within 2 weeks before procedure, (5) with severe liver disease, thyroid dysfunction, malignant carcinoma, or infectious disease. The study protocol conforms to the ethical guidelines of the 1975 *Declaration of Helsinki*. Fuwai Hospital approved this study and waived the requirement for informed consent because of its retrospective design.

Study protocol and definitions

Patients conforming to the standards stated above were included and then divided into CI-AKI and non-CI-AKI group. Clinical characteristics and in-hospital outcomes were extracted from patients' medical records. Procedural characteristics were obtained from laboratory database. Baseline clinical characteristics and medication administration of CI-AKI and non-CI-AKI group were analyzed. Univariable and multivariable logistic analyses were used to identify the risk factors of CI-AKI in emergency PCI patients. For the enrolled cases, treatment strategies and periprocedural medications were based on the protocols of the current guidelines. Hydration started on admission with 1 ml·kg^{-1}·h^{-1} of normal saline and continued until 18–24 h after procedure. In patients with cardiac or renal dysfunction, the volume of hydration was at the physician's discretion. Nonionic, iso-osmolar contrast was administered during the procedure. Serum creatinine (SCr) was measured on admission and daily postprocedure until discharge. CI-AKI was defined as an increase in SCr ≥25% or ≥44.2 μmol/L (0.5 mg/dl) above baseline in 3 days after exposure to contrast medium.[13] The estimated glomerular filtration rate (eGFR) was calculated by CKD-EPI (CKD Epidemiology Collaboration) equation:[14,15] eGFR (ml·min^{-1}·1.73 m^{-2}) = 141 × min (Scr/k, 1)^{a} × max (Scr/k, 1)$^{-1.209}$ × 0.993^{Age} × 1.018 (if female) × 1.159 (if black) where k is 0.7 for females and 0.9 for males, a is −0.329 for females and −0.411 for males, min indicates the minimum of Scr/k or 1, and max indicates the maximum of Scr/k or 1, which was performed by a CKD-EPI calculator on the website (http://www.qxmd.com/calculate-online/nephrology/ckd-epi-egfr).

Statistical analysis

Continuous variables were expressed as median and interquartile range, and Mann–Whitney *U*-test was used to test the statistical difference. Categorical variables were reported as count and percentage, and the difference was tested with Chi-square test or Fisher's exact test. Logistic analysis was performed to identify independent risk factors of CI-AKI. The β coefficient, odds ratio (*OR*), and the corresponding 95% confidence interval (*CI*) were calculated at the same time. A two-sided $P < 0.05$ was considered to indicate statistical significance. All tests were performed by the IBM SPSS Statistics Version 22 statistical software package (SPSS Inc., Chicago, Illinois, USA).

Results

Baseline clinical and procedural characteristics of contrast-induced acute kidney injury and noncontrast-induced acute kidney injury group

A total of 1061 patients were finally included in the study, in which 80.7% (856/1061) were males. The incidence of CI-AKI in emergency PCI patients was 22.7% (241/1061). Baseline clinical characteristics of CI-AKI and non-CI-AKI group are summarized in Table 1. The median ages were 61 years in CI-AKI group and 59 years in non-CI-AKI group with no statistical significance ($P = 0.074$). Females accounted for 27.4% in CI-AKI group, higher significantly than that of non-CI-AKI group ($P < 0.001$). Moreover, CI-AKI patients were prone to higher rate of myocardial infarction (MI) history, lower body surface area (BSA), smoking rate, left ventricular ejection fraction (LVEF) and hemoglobin (Hb) level, higher levels of white blood cell (WBC), platelet, fasting glucose, high-sensitive C-reactive protein (hs-CRP), and eGFR, all variates with significant difference between two groups. Procedural characteristics and medication administration of two groups are shown in Table 2, from which CI-AKI patients were with significantly longer onset-to-balloon time, higher rates of intro-aortic balloon pump (IABP) application, left anterior descending (LAD) impaired, and LAD stented. About periprocedural medication, CI-AKI group was with significantly higher rate of diuretics use and lower rates of aspirin administration. Statistical difference was significant in aspirin use between two groups though there were similarly high percentages (99.9% in CI-AKI group and 98.8% in non-CI-AKI group, respectively). Besides, the rate of patients with contrast volume (CV) >200 ml showed no significant difference between CI-AKI and non-CI-AKI groups.

Univariable analysis for contrast-induced acute kidney injury risk factors in emergency percutaneous coronary intervention patients

Univariate analysis for risk factors of CI-AKI in emergency PCI patients is shown in Table 3. A total of 23 variables were analyzed and 15 variables showed significant association with CI-AKI. The significant correlates included demographics (female [$P < 0.001$] and BSA [$P < 0.001$]), comorbidities

Table 1: Baseline characteristics and laboratory determinations of CI-AKI and non-CI-AKI groups

Variables	CI-AKI (n = 241)	Non-CI-AKI (n = 820)	P
Age (years)	61 (51–69)	59 (51–67)	0.074
Female	66 (27.4)	139 (17.0)	<0.001
BSA (m^2)	1.79 (1.66–1.89)	1.85 (1.73–1.96)	<0.001
Smoking	152 (63.1)	577 (70.4)	0.032
Alcohol	50 (20.7)	192 (23.4)	0.386
Hypertension	162 (67.2)	498 (60.7)	0.052
Hyperlipidemia	203 (84.2)	702 (85.6)	0.596
DM	74 (30.7)	225 (27.4)	0.322
History of MI	51 (21.2)	111 (13.5)	0.004
TIA/stroke	47 (19.5)	120 (14.6)	0.068
SBP (mmHg)	125 (116–140)	123 (114–136)	0.107
DBP (mmHg)	75 (66–82)	72 (64–80)	0.088
LVEF (%)	51 (44–57)	56 (50–60)	<0.001
History of statins	63 (26.1)	186 (22.7)	0.265
WBC ($\times10^9$/L)	10.99 (8.84–13.60)	10.39 (8.32–12.58)	0.009
Hb ($\times10^{12}$/L)	146 (134–155)	147 (137–157)	0.036
Platelet ($\times10^9$/L)	218 (188–259)	214 (179–246)	0.039
Fasting glucose (mmol/L)	7.22 (5.80–9.58)	6.69 (5.57–8.66)	0.005
Triglyceride (mmol/L)	1.40 (1.01–1.94)	1.43 (1.01–1.99)	0.532
Total cholesterol (mmol/L)	4.44 (3.77–5.23)	4.46 (3.85–5.15)	0.913
HDL–c (mmol/L)	1.02 (0.89–1.21)	0.99 (0.85–1.19)	0.132
LDL–c (mmol/L)	2.82 (2.25–3.50)	2.84 (2.27–3.42)	0.851
hs–CRP (mg/L)	9.60 (3.29–11.78)	6.71 (2.86–11.39)	0.002
eGFR (ml·min^{-1}·1.73 m^{-2})	90.9 (74.7–101.8)	85.8 (71.1–96.4)	<0.001

Data are expressed as n (%) or median (interquartile range). CI-AKI: Contrast-induced acute kidney injury; BSA: Body surface area; DM: Diabetes mellitus; MI: Myocardial infarction; TIA: Transient ischemia attack; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; LVEF: Left ventricular ejection fraction; WBC: White blood cell; Hb: Hemoglobin; HDL-c: High-density lipoprotein cholesterol; LDL-c: Low-density lipoprotein cholesterol; hs-CRP: High-sensitive C-reactive protein; eGFR: Estimated glomerular filtration rate.

Table 2: Procedural characteristics and medication administration of CI-AKI and non-CI-AKI groups

Variable	CI-AKI (n = 241)	Non-CI-AKI (n = 820)	P
Onset-to-balloon (h)	8.0 (5.5–12.0)	7.0 (5.0–11.0)	0.019
Door-to-balloon (min)	120 (100–177)	120 (100–170)	0.801
IABP	49 (20.3)	93 (11.3)	<0.001
LAD impaired	220 (91.3)	685 (83.5)	0.003
LAD stented	142 (58.9)	300 (36.6)	<0.001
Volume of contrast medium >200 ml	20 (8.3)	42 (5.1)	0.065
Aspirin	238 (98.8)	819 (99.9)	0.039
Clopidogrel	241 (100)	818 (99.8)	1.000
ACEI	189 (78.4)	633 (77.2)	0.688
ARB	20 (8.3)	68 (8.3)	0.998
β–blocker	212 (88.0)	702 (85.6)	0.352
CCB	14 (5.8)	58 (7.1)	0.493
Diuretics	156 (64.7)	298 (36.3)	<0.001

Data are expressed as n (%) or median (interquartile range). CI-AKI: Contrast-induced acute kidney injury; IABP: Intro-aortic balloon pump; LAD: Left anterior descending; ACEI: Angiotensin-converting enzyme inhibitor; ARB: Angiotensin II receptor blocker; CCB: Calcium channel blocker.

indexes (history of MI [P = 0.004], LVEF [P < 0.001], Hb [P = 0.016], and eGFR [P = 0.005]), risk factors for CAD (smoking [P = 0.032] and fasting glucose [P = 0.009]), inflammation factors (WBC [P = 0.003] and hs-CRP [P = 0.001]), procedural characteristics (IABP [P < 0.001], LAD impaired [P = 0.003], and LAD stented [P < 0.001]), and medication administration (aspirin [P = 0.044] and diuretics use [P < 0.001]).

Logistic multivariable analysis for contrast-induced acute kidney injury risk factors in emergency percutaneous coronary intervention patients

Logistic multivariable analysis of CI-AKI and non-CI-AKI groups in emergency PCI patients is shown in Table 4. A total of eight variables showed significant difference which were BSA (*OR* 0.213, 95% *CI*: 0.075–0.607, P = 0.004), history of MI (*OR* 1.642, 95% *CI*: 1.079–2.499, P = 0.021), LVEF (*OR* 0.969, 95% *CI*: 0.944–0.994, P = 0.015), Hb (*OR* 0.988, 95% *CI*: 0.976–1.000, P = 0.045), eGFR (*OR* 1.027, 95% *CI*: 1.018–1.037, P < 0.001), LAD stented (*OR* 1.464, 95% *CI*: 1.000–2.145, P = 0.050), aspirin use (*OR* 0.097, 95% *CI*: 0.009–0.987, P = 0.049), and diuretics use (*OR* 1.850, 95% *CI*: 1.233–2.777, P = 0.003), being the independent predictors of CI-AKI in emergency PCI patients.

Discussion

CI-AKI has become a serious complication with wide application of PCI technology in CAD patients. From previous studies, a strong correlation has been identified

Table 3: Univariable analysis for CI-AKI risk factors in emergency PCI patients

Variable	β	*OR*	95% *CI*	*P*
Age	0.010	1.010	0.998–1.023	0.100
Female	0.614	1.848	1.319–2.588	<0.001
BSA	−1.838	0.159	0.070–0.362	<0.001
Smoking	−0.330	0.719	0.532–0.972	0.032
Hypertension	0.282	1.326	0.979–1.796	0.068
History of MI	0.539	1.715	1.186–2.478	0.004
TIA/stroke	0.346	1.413	0.973–2.052	0.069
SBP	0.006	1.006	0.998–1.015	0.150
DBP	0.010	1.011	0.999–1.022	0.066
LVEF	−0.070	0.932	0.915–0.949	<0.001
WBC	0.062	1.064	1.021–1.109	0.003
Hb	−0.011	0.989	0.981–0.998	0.016
Platelet	0.002	1.002	1.000–1.004	0.053
Fasting glucose	0.056	1.057	1.014–1.102	0.009
hs-CRP	0.056	1.058	1.024–1.092	0.001
eGFR	0.011	1.011	1.003–1.019	0.005
Onset-to-balloon	0.007	1.007	0.993–1.020	0.331
IABP	0.691	1.995	1.363–2.920	<0.001
LAD impaired	0.725	2.065	1.272–3.351	0.003
LAD stented	0.911	2.486	1.854–3.334	<0.001
Volume of contrast medium >200 ml	0.517	1.676	0.964–2.914	0.067
Aspirin	−2.334	0.097	0.010–0.936	0.044
Diuretics	1.168	3.215	2.381–4.340	<0.001

β: Logistic correlation coefficient; *OR*: Odds ratio; 95% *CI*: 95% confidence interval; CI-AKI: Contrast-induced acute kidney injury; PCI: Percutaneous coronary intervention; BSA: Body surface area; MI: Myocardial infarction; TIA: Transient ischemia attack; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; LVEF: Left ventricular ejection fraction; WBC: White blood cell; Hb: Hemoglobin; hs-CRP: High-sensitive C-reactive protein; eGFR: Estimated glomerular filtration rate; IABP: Intro-aortic balloon pump; LAD: Left anterior descending.

Table 4: Multivariable logistic analysis for CI-AKI risk factors in emergency PCI patients

Variable	β	*OR*	95% *CI*	*P*
Female	0.190	1.209	0.728–2.008	0.464
BSA	−1.548	0.213	0.075–0.607	0.004
Smoking	−0.064	0.938	0.640–1.374	0.742
History of MI	0.496	1.642	1.079–2.499	0.021
LVEF	−0.032	0.969	0.944–0.994	0.015
WBC	0.035	1.036	0.986–1.087	0.158
Hb	−0.012	0.988	0.976–1.000	0.045
Fasting glucose	0.033	1.034	0.986–1.084	0.167
hs-CRP	0.030	1.031	0.994–1.069	0.097
eGFR	0.027	1.027	1.018–1.037	<0.001
IABP	0.264	1.302	0.824–2.056	0.258
LAD impaired	0.188	1.207	0.702–2.075	0.496
LAD stented	0.381	1.464	1.000–2.145	0.050
Aspirin	−2.336	0.097	0.009–0.987	0.049
Diuretics	0.615	1.850	1.233–2.777	0.003

β: Logistic correlation coefficient; *OR*: Odds ratio; 95% *CI*: 95% confidence interval; CI-AKI: Contrast-induced acute kidney injury; PCI: Percutaneous coronary intervention; BSA: Body surface area; MI: Myocardial infarction; LVEF: Left ventricular ejection fraction; WBC: White blood cell; Hb: Hemoglobin; hs-CRP: High-sensitive C-reactive protein; eGFR: Estimated glomerular filtration rate; IABP: Intro-aortic balloon pump; LAD: Left anterior descending.

between CI-AKI and adverse clinical outcome, especially in ACS patients.[6,10,16] The emergency cases should be paid more attention due to high rate of CI-AKI and limited prophylactic strategies.[17] Whereas, the reported risk factors for CI-AKI were mostly based on data from selective procedure patients with stable systemic condition and adequate preparation before procedure, which might be improper to be used in those undergoing emergency PCI in clinical practice. In the body of the literature, we explored the risk factors of CI-AKI postemergency PCI in a Chinese population.

In the present study, the incidence of CI-AKI was 22.7% in patients undergoing emergency PCI, higher than previous studies.[2,3] From the results of statistical analysis, the independent predictors of CI-AKI in emergency PCI patients included 8 readily available variables which were patient-related (BSA, history of MI, LVEF, Hb, and eGFR), procedure-related (LAD stented), and medication-related (aspirin and diuretics use) variables.

The pathogenesis behind CI-AKI has not been completely understood. Multiple mechanisms may be involved which are sustained intrarenal vasoconstriction, direct cytotoxic effect of contrast medium, renal medullary hypoxia, ischemic injury, oxidative stress, and inflammation.[4] In the study, history of MI, LVEF, and LAD stented were independent predictors for CI-AKI postemergency PCI, same results to prior study,[18] showing that cardiac function plays a vital effect on CI-AKI in both selective and emergency PCI patients. The possible mechanism may be that cardiac dysfunction perturbs renal hemodynamics affecting renal tubulodynamics and then leading to regional hypoxia.[4] Moreover, due to the similar mechanism of hypoxia, lower Hb level was regarded as an independent risk factor of CI-AKI in the present study, supporting Mehran *et al*.'s report.[18] In addition, a low BSA was shown as an independent risk factor of CI-AKI for the first time in our study. BSA, a new variable in the risk factor profile of CI-AKI, is a body size index usually used as the ratio of CV/BSA to show a high specificity in prediction of CI-AKI compared to a single CV variate.[18] Kondo *et al*.[19] concluded that BSA was a simple and feasible index for the determination of iodine dose in individual patients. With the fact that toxicity intensity of contrast medium is related to both CV and BSA, and that there was no statistical significance in the rate of patients with CV >200 ml in the body of the literature which was due to the popularization of nonionic, iso-osmolar contrast medium and the lowest volume used during intervention procedure in recent years, BSA, representing the area where drug distributes, showed the toxicity intensity of contrast medium.[20] In other words, BSA was an alternate of CV in the risk factor profile of CI-AKI in emergency procedure population in the study.

As for the periprocedural medication, the administration of aspirin could reduce the risk of CI-AKI from the study though only three and one patient did not contact aspirin in CI-AKI and non-CI-AKI group, respectively, supporting the

hypothesis that contrast medium could induce thrombosis in the development of CI-AKI.[21] Aliev *et al*.'s study[21] also clarified that administration of heparin before procedure could significantly decline the rate of CI-AKI, indicating that additional antithrombotic treatment may matter a lot in CI-AKI prevention. However, the use of diuretics could increase the risk of CI-AKI, similar to Solomon *et al.*'s conclusion.[22] The occurrence of CI-AKI is related to toxic effect of contrast medium on the tubular epithelial cells and results directly from hemodynamic disturbances of the renal blood flow.[23] The pre- or post-procedural use of diuretics can directly lead to CI-AKI through reducing renal blood flow and enhancing the toxicity of contrast medium due to blood concentration. By contrast, periprocedural hydration, as the cornerstone of CI-AKI prevention, can help reverse the negative hemodynamic conditions in clinical practice.[13]

In the body of the literature, some clinical factors were inconsistent with previous conclusions. A higher eGFR served as a promoting factor in the study showing contrary performance to prior reports,[18,24,25] which might be attributed to high sensitivity of CI-AKI definition and special nature of the study population. A higher eGFR usually came from a relatively lower SCr correspondingly and was apt to be diagnosed as CI-AKI for a slight fluctuation but SCr increases ≥25% above baseline. As a fact, a small fluctuation is easily occurring among emergency PCI patients. Consequently, eGFR showed the illusion of positive correlation with CI-AKI through CKD-EPI formula conversion. Besides, preprocedural statin contact showed no statistical significance in the present study, while several previous studies[26-29] concluded that statin therapy was effective in reducing the risk of CI-AKI in ACS patients owing to pleiotropic effects (the anti-inflammatory, anti-apoptotic, and anti-thrombotic properties) with few side effects. The probable reasons of contrary results might be attributed to the paucity of high-dose statin cases, small duration of statin contact before procedure, and diversification of statin administration in the real word. In future, large and well-designed studies are needed to confirm the preventive effect of preoperative statin therapy and determine the rational dose and timing in emergency patients.

Above all, comparing to prior studies, low BSA and diuretics use are two new risk factors of CI-AKI in our study. Moreover, some identified risk factors in previous reports are also present in our research, for example, low LVEF, Hb level, and LAD stented. Whereas, some other variables, such as age, female, hypertension, diabetes mellitus, peripheral vascular disease, IABP application, and CV, were reported to be independent risk factors of CI-AKI in selective PCI cases,[11,18,30,31] showing no significance in our study. Hence, different kinds of patients should deserve different risk factor profile to achieve the most accurate estimation of the risk of CI-AKI clinically.

There are several limitations. First, the present study was based on patients enrolled from a single center and the data were collected retrospectively. Therefore, our results are subjected to limitations inherent to the observational nature of a retrospectively collected database. Second, due to the regular application of periprocedural hydration, we could not confirm the concrete and specific influence exerted on the true baseline SCr before intervention. Third, though the definition of CI-AKI in the present study is used universally in previous reports, it might not be suitable for emergency population according to eGFR's abnormal performance. A more scientific and appropriate critical value of SCr increase in the definition of CI-AKI in an emergency PCI population should be explored in future.

This study demonstrated that the incidence of CI-AKI in emergency PCI patients was high. History of MI, low BSA, LVEF and Hb level, LAD stented, and diuretics use are associated with increased risk of CI-AKI in patients undergoing emergency PCI.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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