Original Article

Effect of sodium bicarbonate infusion in off-pump coronary artery bypass grafting in patients with renal dysfunction

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Abstract

Background and Aims: Acute kidney injury (AKI) following cardiac surgery is a major complication resulting in increased morbidity, mortality and economic burden. This study was designed to determine the benefit of sodium bicarbonate ($NaHCO_3$) supplementation in patients with stable chronic kidney disease (CKD) undergoing off-pump coronary artery bypass grafting (OP-CABG).

Material and Methods: We prospectively studied 60 non-dialysis CKD patients with glomerular filtration rate (GFR) ≤60 ml/min/1.73 m^2 requiring elective OP-CABG. They were randomly allocatted to one of the two groups. One group received $NaHCO_3$ infusion at 0.5 mmol/kg first hour followed by 0.2 mmol/kg/h till the end of surgery and the other group received 0.9% NaCl. A third group of 30 patients without renal dysfunction undergoing OP-CABG was included. The serum creatinine was estimated prior to surgery, immediately after surgery and on postoperative days 1, 2, 3 and 4.

Results: Ten patients (33.3%) in NaCl and 6 (20%) patients each in $NaHCO_3$ and normal groups developed Stage-1 AKI. None of our study patient required renal replacement therapy and no mortality was observed in any of the groups during the perioperative and hospitalization period.

Conclusion: Perioperative infusion of $NaHCO_3$ in OP-CABG reduced the incidence of Stage-1 AKI by about 40% when compared to NaCl. The incidence of Stage-I AKI in $NaHCO_3$ group was similar to that in patients with normal renal function undergoing OP-CABG. A larger group of patients may be required to suggest a significance of renal protective benefit of $NaHCO_3$ in patients undergoing OP-CABG.

Keywords: Acute kidney injury, cardiac surgery, coronary artery bypass grafting, creatinine, sodium bicarbonate

Introduction

Acute kidney injury (AKI) after cardiac surgery continues to be a major devastating complication because it may result in multi-organ dysfunction, death, increased resource utilization and high cost.[1] Globally, 800,000 patients undergo coronary revascularization annually with the use of cardiopulmonary bypass (CPB). Approximately 77,000 patients in a year develop postoperative AKI, among which 14,000 require dialysis for the first time.[2] Up to 30% of patients undergoing coronary artery bypass grafting (CABG) sustain sufficient renal injury to meet the threshold criteria, i.e., a creatinine increase of >0.3 mg% or 50% of their baseline within 48 h of surgery. The reported incidence of AKI after cardiac surgery varies according to the definition of kidney injury as well as the institution reporting the results. Additionally, the current models poorly predict the likelihood of AKI. About 3% of

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patients sustain AKI following CABG of sufficient severity requiring dialysis. As many as 60% of patients requiring dialysis after CABG die before hospital discharge and the survivors continue with chronic renal disease with or without the need for dialysis.

Serum creatinine reflects the balance between the synthesis of creatinine and its excretion by the kidney. Creatinine production in the body varies with muscle mass, physical activity, protein intake and catabolism while creatinine excretion is dependent on the glomerular filtration rate (GFR). The serum creatinine and GFR are inversely and exponentially related. Halving of GFR implies that there will be doubling of creatinine concentration.[3] Maintenance of volume status along with pretreatment with sodium bicarbonate ($NaHCO_3$) has been shown to be effective for prophylaxis for contrast-induced nephropathy.[4] Alkalization reduces free radicals responsible for renal injury[5-8] and pretreatment with $NaHCO_3$ has been demonstrated to be more protective than sodium chloride (NaCl) in both doxorubicin and ischemia-induced animal models of acute renal failure.[9-11] In a small, randomized, double-blind trial of 100 patients undergoing cardiac surgery who were at increased risk of AKI, a 24 h infusion of $NaHCO_3$ decreased the incidence of acute renal dysfunction.[12] The effects of $NaHCO_3$ infusion in patients with coronary artery disease (CAD) with preexisting renal dysfunction (non-dialysis dependent) has not been studied in patients undergoing OP-CABG to the best of our knowledge.

Present study was designed to determine the effect of urinary alkalization with systemically administered $NaHCO_3$ in those patients who have preexisting renal dysfunction but not on maintenance dialysis and scheduled for OP-CABG.

Material and Methods

We prospectively studied 60 adult (aged 18 years or above) consecutive stable chronic kidney disease (CKD) patients not on dialysis who were scheduled for elective off-pump CABG (OP-CABG) at a tertiary care cardiac center. This study was approved by the ethics committee and institutional review board. All these patients were identified by eGFR ≤60 ml/min/1.73 m^2 or creatinine ≥1.4 mg% prior to OP-CABG. Preoperative eGFR was estimated by using modification of diet in renal disease (MDRD) formula.

We excluded patients scheduled for on-pump CABG, emergency surgery or redo operations, and those who had end-stage renal disease, chronic inflammatory disease or immune-suppression, those enrolled in a conflicting research study, and patients on corticosteroid therapy, renal replacement therapy (RRT) and renal-transplanted patients. All patients received a standard anesthetic consisting of midazolam, isoflurane, fentanyl, vecuronium or atracurium, endotracheal intubation and mechanical ventilation adjusted to achieve normocarbia. Following median sternotomy and heparinization, distal coronary anastomosis was done on the beating-heart using "octopus" (Medtronic Inc, Minneapolis, MA, USA) suction device tissue stabilizer for immobilization of the local heart muscle. Those requiring conversion to "on-pump" (CPB) were excluded from the study.

Surgical procedure

Hemodynamic management included a targeted mean arterial pressure of at least 70 mmHg, central venous pressure of 8–12 mmHg, pulse pressure variation (PPV) of ≤12% using either Flotrac (Vigileo, I PX 1, Edward Lifesciences, Irvine, USA) or Lidco rapid (model POC-125, ADVANTECH, Taiwan) when not on intra-aortic balloon plasty (IABP) and cardiac index of ≥2.5 L/min/m^2. Infusion of epinephrine at a rate of 0.01–0.05 μg/kg/min and nitroglycerine at a rate of 0.05–0.1 μg/kg/min were used as inotrope/vasodilator at the discretion of the anesthesia care team. All patients were electively ventilated postoperatively until the criteria for separation from ventilator and tracheal extubation were met. Postoperative analgesia was provided with fentanyl infusion at 0.5–1.0 μg/kg/h until removal of chest tube/s and then only paracetamol. No nephrotoxic agents were used and non-steroidal anti-inflammatory drugs were avoided in all patients.

Patients were randomly allocated to one of the two groups to receive $NaHCO_3$ at a rate of 0.5 mmol/kg/h during the first hour of surgery followed by 0.2 mmol/kg/h[13,14] till the end of surgery and standard care or 0.9% NaCl infusion instead of $NaHCO_3$ and standard care. In addition, simultaneously we included age-matched control group of 30 patients who did not have any renal dysfunction and were undergoing OP-CABG with a preoperative serum creatinine of ≤1.4 mg/dL and eGFR ≥60 ml/min/1.73 m^2. Anesthesia and surgery were similar in all the three groups, mean arterial pressure and intravascular volume status was maintained with appropriate clinical measures during the course of surgery in all patients. Blood samples were drawn at specified intervals namely, at the beginning of surgery after anesthetic induction, at the end of surgery and 24 h after surgery, for measurement of serum creatinine, troponin-T, and prior to, at the end of surgery and at 24 h after surgery. The diagnosis of postoperative AKI was made using "kidney

disease; improving global outcomes" (KDIGO) criteria[15] in terms of increase in serum creatinine on any of the first five postoperative days. RRT was instituted based on the criteria listed in Table 1.

CABG-related myocardial infarction (MI) was defined as elevation of cardiac biomarker, i.e., troponin-C values ($\leq 10 \times 99^{th}$ percentile URL) in patients with normal baseline cTn values along with either (i) new pathological Q waves or new left bundle branch block (LBBB) or (ii) angiographic documented new graft or new native coronary artery occlusion, or (iii) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

Statistical analysis

The data were analyzed by the SPPS (Version 15, SPSS Inc., Chicago, USA) and Microsoft excel. Standard statistical methods for assessment on proportions, percentages and measures of central tendencies (mean, SD) were used. One-way analysis of variance (ANOVA) was used to determine the significance of variables between the groups.

Results

Basic demographics and clinical data of each group are described in Table 2. By design, the preoperative serum creatinine was significantly higher and eGFR significantly lower in the NaCl and $NaHCO_3$ groups compared to the normal group. The numerical Euroscore was higher in the NaCl and $NaHCO_3$ groups compared to the normal group. Prevalence of diabetes mellitus and hypertension was similar in all the three groups. Two patients, one in normal group and one in $NaHCO_3$ were converted to on-pump due to surgical reasons. One patient in each NaCl group and normal group needed IABP for cardiac support. The incidence of further deterioration in renal function postoperatively is described in Table 3. None of the patients required re-exploration for bleeding or prolonged ventilation. All patients were discharged from intensive care unit and hospital in a hemodynamically stable condition. Six patients (20%) in normal control group, 10 (33.3%) patients in NaCl group and 6 (20%) patients in $NaHCO_3$ developed Stage-1 AKI. However, there was no incidence of Stage-2 and Stage-3 AKI in any of the group. The elevation of serum creatinine in each group is is shown in Table 3. There was no significant difference in other variables with infusion of $NaHCO_3$ as compared to NaCl group [Table 3].

Table 1: Criteria used for initiating renal replacement therapy during the postoperative period

Condition	Description
Oliguria	≤200 ml/12 h
Anuria	Urine output 0-50 ml/12 h
Blood urea	≥35 mmol/L or ≥98 mg/dL
Serum creatinine	≥400 mmol/L or ≥4.5 mg/dL
Uncompensated metabolic acidosis	pH ≤7.1
Serum potassium	≥6.5 mmol/L or rapidly rising values
Serum sodium	≤110 and ≥160 mmol/L
Pulmonary edema	Unresponsive to diuretics
Uremic manifestations	Encephalopathy

Discussion

AKI after cardiac surgery is not an uncommon complication. AKI is associated with increased in morbidity, mortality and hospital costs. Even when RRT (dialysis) is avoided, milder forms of AKI are associated with adverse outcomes and degree of AKI correlates negatively with long-term survival despite successful hospital discharge. Renal dysfunction after cardiac surgery is multifactorial in origin and there are multiple risk factors that contribute to the development of AKI.

Both the study groups (placebo and sodium bicarbonate) had preexisting renal dysfunction. But none required RRT, and none demonstrated Stage-2 or Stage-3 AKI following the surgery. There was a 40% reduction in incidence of Stage-1 AKI in patients who received $NaHCO_3$ compared to NaCl group in patients who suffered from preexisting renal dysfunction. However, the incidence of Stage-1 AKI

Table 2: Demographic and clinical data of patients undergoing off-pump coronary artery bypass grafting in the study

Variable	Normal control[§] (*n*=30)	NaCl group[#] (*n*=30)	$NaHCO_3$ group[¶] (*n*=30)	*P* (one-way ANOVA)
Age (years)	57.11±9.5	60.87±7.1	60.81±9.2	NS
Male/female	28/2	27/3	29/1	NS
Diabetes mellitus (%)	17 (56)	15 (50)	14 (46)	NS
Hypertension (%)	17 (56)	18 (60)	15 (50)	NS
Euro score	2.30±1.97	4.48±2.39**	4.67±2.22**	<0.001
Preoperative eGFR (ml/min/1.73 m^2)	84.65±16.63	49.05±10.89**	50.44±7.17**	<0.001
Preoperative creatinine (mg%)	0.99±0.15	1.43±0.15**	1.54±0.33**	0.001

*[§]Patients with normal renal function; [#]patients with preexisting renal dysfunction and received sodium chloride; [¶]patients with preexisting renal dysfunction and received sodium bicarbonate; One-way ANOVA test, **P<0.001 for between group comparison; NS=Not significant; eGFR=Estimated glomerular filtration rate*

Table 3: Creatinine, urine output and troponin data of study patients

Variable	Normal control group[§] (*n*=30)	NaCl group[#] (*n*=30)	$NaHCO_3$ group[¶] (*n*=30)	*P*[¶¶]
Creatinine (mg%) mean±SD				
Immediate preoperative	0.99±0.16	1.56±0.43**	1.48±0.41**	<0.001
Immediate postoperative	1.01±0.27	1.60±0.47**	1.49±0.42**	<0.001
Postoperative 24 h	1.04±0.16	1.62±0.52**	1.45±0.48*	<0.05
Postoperative 48 h	1.03±0.17	1.64±0.52**	1.45±0.59*	<0.05
Postoperative 72 h	0.96±0.15	1.53±0.35*	1.42±0.53	<0.05
Postoperative 96 h	0.97±0.14	1.52±0.35*	1.43±0.49	<0.05
Troponin-1 (ng/ml) (mean±SD)				
Preoperative	0.04±0.1	0.18±0.4	0.07±0.1	NS
Immediate postoperative	2.15±6.2	5.69±12.5	8.57±16.6	<0.01
Postoperative 24 h	6.71±15.2	3.69±5.6	20.74±44.4	<0.05
Stage-1 AKI (N)[$$]	6	10	6	
Urine output (ml/24 h)				
Day 1 postoperative	2183.86±658.94	2066.57±629.78	2479.28±581.81	
Day 2 postoperative	3184.77±786.57	3299.47±405.31	2820.85±897.78	
Day 3 postoperative	3101.25±672.91	2664.41±792.13	2881.25±928.06	

*[§]Patients with normal renal function; [#]patients with preexisting renal dysfunction and received sodium chloride; [¶]patients with preexisting renal dysfunction and received sodium bicarbonate; [$$]serum creatinine elevation by 0.3 mg%; [¶¶]one-way ANOVA test, *P<0.05; **P<0.001 for between group comparison; NS=Not significant; SD=Standard deviation; AKI=Acute kidney injury*

was similar in patients received $NaHCO_3$ and patients with normal renal function. This is suggestive of possible renal protective effect of $NaHCO_3$ infusion in patients undergoing OP-CABG with preexisting renal dysfunction.

In a meta-analysis with total of 1092 patients, the influence of alkalinization of urine with $NaHCO_3$ was studied in patients undergoing cardiac surgery.[16] The incidence of AKI, requirement for RRT, duration of postoperative mechanical ventilation, length of ICU/hospital stay and death was determined using accepted methods. Though the treatment methodology varied a little in each of the studies, $NaHCO_3$ was infused at a dose 0.5 mmol/kg/h for the first hour followed by 0.2 mmol/kg/h infusion.[13,14]

There was no significant difference in the occurrence of AKI, need for RRT, length of stay in ICU and hospital mortality in the two groups namely with $NaHCO_3$ and without $NaHCO_3$.[16] Hemolysis is relatively common in extracorporeal circulation which may predispose patients to AKI. Urinary acidity may enhance generation and toxicity of reactive oxygen species. In addition, activation of complement during cardiac surgery may precipitate AKI. Urinary alkalinization may protect the kidney from AKI induced by oxidant substrates, iron-mediated free radicals and tubular cast formation. In addition, urinary alkalinization could prevent generation of hemoglobin casts, inhibit endocytic hemoglobin uptake and attenuate tubular necrosis. Alkalinization of urine based on $NaHCO_3$ infusion therapy has been reported to benefit patients with contrast-induced nephropathy.[4] This prospective study showed the beneficial effect of $NaHCO_3$ infusion in patients with stable CKD (with GFR ≤60 ml/min/1.73 m^2) undergoing OP-CABG. The fundamental issue in the maintenance of renal integrity revolves round maintenance of oxygen supply and demand especially to the renal medulla. The factors which maintain this supply-demand balance are the mean arterial pressure during CPB and the oxygen-carrying capacity. Many authors have proposed renal protective strategies to prevent AKI or at least detect deterioration in the renal function in a timely manner to permit early intervention while evaluating genetic influences that may predispose the patients to AKI during cardiac surgery.

Our study findings disagree with the results of another study reported recently by Kristeller *et al.* However, this prospective randomized study was done in patients undergoing cardiac surgery under cardiopulmonary bypass.[17] On the other hand, in a prospectively planned double-blind randomized controlled trial looking at $NaHCO_3$and renal function after cardiac surgery with individual patient data meta-analysis (IPDMA) on 877 patients, urinary alkalization using $NaHCO_3$ did not decrease the overall incidence of AKI, but it reduced severe AKI and need for RRT in low-risk elective CABG.[18] Our study produced similar results showing a trend towards a reduction in Stage-1 AKI with $NaHCO_3$ infusion. The avoidance of CPB may have also played a role in influencing renal outcomes but is unlikely as shown by a recent report by Hynes and associates.[19]

One recent study demonstrated that periprocedural intravenous isotonic $NaHCO_3$ showed no benefit over intravenous isotonic

NaCl with respect to the risk of major adverse kidney events, death or AKI, in patients undergoing angiography.[20]

A double-blind randomized control study by Soh *et al.*, reported that perioperative administration of $NaHCO_3$ does not prevent the postoperative AKI in after off-pump coronary revascularization. The study showed similar incidences of AKI in $NaHCO_3$ group and control group who received same amount of 0.9% NaCl (21% vs 26%; $P = 0.458$), and there were no differences in proportions of AKI stages.[21] However, in contrast to this study, we found that perioperative administration of $NaHCO_3$ reduced the incidence of postoperative AKI in patients with preexisting renal dysfunction and underwent OP-CABG. This may be attributed to the methodological differences between the two studies, e.g., patients with an estimated GFR of 30–89 ml/min/1.73 m^2 were grouped as preexisting chronic renal disease in Soh's study where as our study considered eGFR of <60 ml/min/m^2. Secondly balanced hydroxyethyl starch (HES) 130/0.4 solution was used to compensate for blood loss in Soh's study but no HES was used in our study.

Conclusion

Our current study findings show that the perioperative administration of $NaHCO_3$ infusion in OP-CABG reduces the incidence of Stage-1 AKI in patients with preexisting renal dysfunction. However, a study on larger group of patients required to strengthen our findings.

Limitations of the study

One of the important limitations of our study is small number of patients ($n = 90$) tested.

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Conflicts of interest

There are no conflicts of interest.

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CONFERENCE CALENDAR July-Sepetember 2018

Name of conference	Dates	Venue	Name of organising Secretary with contact details
IAPA 2019 11th Annaul national Conference of Association of Pediatric Anaesthesiologists	8th-10th February 2019	AIIMS, new Delhi	Prof Rajeshwari Prof & Head Deptt of Anesthesiology & Critical care Conference Secretariat Address : Room no. 5014 A, 5th floor Teaching Block, Ansari Nagar, AIIMS New Delhi – 110029 Dr. Manpreet : +91 9868595487 Dr. Anjolie Chhabra : +91 9810104383 Email : iapaaiims2019@gmail.com
ISNACC 2019	15th-17th February 2019	Gurugram India	www.isnacc2019.com
22nd IACTACON 2019 22nd Annual National Conference of Indian Association of Cardiovascular Thoracic Anaesthesiologists	22nd -24th February 2019	Swabhumi The Heritage, Kolkata, West Bengal, India	Dr. Rahul Guhabiswas Org Secretary CIMGlobal India Pvt. Ltd BB – 31, Ground Floor, Salt Lake City, Sector – I Near Punjab National Bank, Kolkata – 700064 Mr. Gaurav Sinha Email: gaurav@cimglobal.in iacta2019@gmail.com
29th Annual National Conference of the Research Society of Anaesthesiology Clinical Pharmacology	29th -31st March 2019	PGIMS , Rohtak	Prof Dr Savita Saini Prof & Head, Deptt of Anesthesiology & Critical Care, PGIMS, Rohtak Email: www.rsacpcon2019@gmail.com Website: www.rsacpcon2019.com