

ANALYZE THE IMPACT OF ALTERNATIVE ROUTES AND TIMING OF DOPAMINE AND MANNITOL ADMINISTRATIONS TO REDUCE NEGATIVE PROPERTIES OF EXTENDED CARDIOPULMONARY BYPASS ON RENAL FUNCTION IN CORONARY ARTERY OPERATIONS

ABSTRACT

Objectives: To analyze impact of alternative routes and timing of dopamine and mannitol administrations to reduce negative properties of extended cardiopulmonary bypass on renal function in coronary artery operations.

Methods: Set I (n: 26 individual): Mannitol (1 g/kg) has been introduced to the CPB priming solution. Set II (n: 25 patients): Even during interval among anesthetic induction and operation, 3 g/kg/min of IV dopamine was delivered. Group III (n = 25 patients): 2 g/kg/min IV dopamine was provided among anesthesia initiation and operation conclusion, and 1 g/kg mannitol were added to priming solution for CPB. Furosemide was administered to Group IV (n = 26 cases) when urine production was poor.

Results: There would be a substantial rise in the post-operative urine microalbumin/creatinine ratios over all classes (p 0.06), as well as a rise in cystatin-c in Set 1, 2, and 3 (p 0.02).

Conclusions: Researchers suggest that combining dopamine infusion (1 g/kg/min) and mannitol (2 g/kg) throughout CPB seems to be the more actual method for preventing detrimental possessions of CPB on renal functioning.

KEY WORDS: Alternative Routes, Pharmacological Protection, Renal Function, Coronary artery surgery.

INTRODUCTION

Renal function is compromised in 32-44 percent of individual subsequent coronary artery bypass grafting surgery, through 2 to 4% experiencing acute renal letdown [1]. A variety of etiological causes remain to blame for decrease in renal perfusion and the ischemia damage. For example, throughout cardiopulmonary bypass, an increase in renal vascular impedance remains associated by the 34% decrease in renal blood flow. Even in context of supportive therapy such as hemodialysis and high dosage inotropic support, ARF has substantial morbidity and death rates [2].

Reduced dopamine (2-4 g/kg/min) works via interacting with dopaminergic receptors in renal arteries (i.e., DA1 and DA2). Dopamine similarly raises cardiac output, preserves renal perfusion, lowers renal metabolism, in addition causes diuresis [3].

Mannitol treatment after CPB permits glomerular capillary pressure to be maintained through avoiding tubular blockage. This similarly lowers plasma stages of hydrogen peroxide permitted oxygen radicals, decreases ischemia-related protein leak from renal vasculature, and protects the kidneys from harm [4].

Our study seeks to assess the impact of proposed routes and timings of dopamine and mannitol management, that were utilized to mitigate negative belongings of extended CPB and to protect renal function, by analyzing multiple renal function measures [5].

METHOS

This research comprised 100 ASA III individuals having normal kidney function, EF more than 43%, and adequate protein also electrolyte levels which had been arranged for elective CABG surgery at Punjab Institute of Cardiology in Lahore. The hospital's independent commission accepted the study plan. In addition, each patient provided informed consent.

Prohibiting criteria comprised use of ACE inhibitors or diuretics, a fresh myocardial infarction, anemia, diabetes, in addition the usage of radiological contrast material during previous three days. Urine microalbumin, urinary creatinine, and serum cystatin-c levels remained evaluated on preoperative and postoperative Day 2.

Following 100% oxygen inhalation, 2 mg/kg propofol, 15 g/kg/min fentanyl, and a bolus intravenous (IV) 0.2 mg/kg alcuronium were administered for anesthesia induction. Urine output has been measured hourly using the urinary flow measurement equipment and a urinary bladder catheter implanted afterward intubation (Bicircular Uri meter 500, sterile closed urine dimension system=refix). And per the manner of dopamine and mannitol administration, participants were divided into two clusters:

Group I (n:25 patients): Mannitol (1 g/kg) remained extra to CPB priming solution. Mannitol was still not added to the priming solution in Sample 2 (n=26 patients). Following anesthetic induction, IV dopamine

remained delivered at the rate of 2 g/kg/min till operation was completed. Group 3 (n:26 patients): Following anesthetic induction, IV dopamine remained managed at the rate of 2 g/kg/min till operation was completed. Mannitol at a concentration of 1 g/kg was added to the priming solution. Class IV (n:27 individuals) (Controls): No dopamine or mannitol were added to priming solution, and patients with poor urine output were provided furosemide. Other than during CPB, a mean arterial pressure of 68 to 100 mmHg and a heart rate of 63 to 100 beats per minute were desired. In hypotensive individuals, rapid volume replacement remained undertaken. Whenever it failed to reestablish target blood pressure levels, ephedrine was administered. For hypertensive episodes, 1-3 g/kg/min nitroglycerine remained utilized. For cardiac protection, standard cardiac operation cannulation has been done, and mild hypothermia and blood cardioplegia remained employed. For CPB, a Biomedicals pump system and a membrane oxygenator (Di deco 0.709 Simplex) remained used.

In Groups 1 and 3, pumping priming solution included lactated Ringer's (30 ml/kg) + Heparin (1 mg/kg) + Mannitol 23 percent (1 g/kg) in addition to 60 ml of NaHCO₃. The hematocrit number was controlled among 21 and 27 percent during CPB (i.e., hemodilution). Heparin at a dosage of 3-5 mg/kg remained supplied for systemic heparinization based on initial enabled coagulation time, that remained kept overhead 420 sec having 35-minute ACT nursing periods. A MAP of 50 to 100 mmHg remained aimed for hemodynamic stabilization in critical care unit. Blood transfusions have been administered to keep the hematocrit level over 27%.

Statistical analyses: For statistical studies, SPSS for Windows version 17.0 was utilized. In addition to descriptive statistical approaches, the one-way Anova test was employed as the parametric test in among comparison, and the Tukey HSD test was utilized to determine which group was directly proportional to the difference (mean, standard deviation). The Kruskal Wallis test remained utilized as the non-parametric test among

group comparison. In inside comparisons, the paired sample t trial remained employed as the parametric test, while the Wilcoxon sign assessment remained utilized as the non-parametric test. The Chi-square assessment remained employed to compare qualitative data. The statistical analyses were performed at the 96 percent confidence level, with the p level of less than 0.05 regarded statistically substantial.

UNDER PEER REVIEW

Table-I: Demographic features of sets.

	Set 1	Set 2	Set 3	Set 4	p
	Mean±SD	Mean±SD	Mean±SD	Mean±SD	
Age	58.37±12.12	58.57±9.38	57.65±10.11	58.57±8.01	0.731
Height	166±5.2	168±9.7	68±5.3	169±7.2	0.881
Body weight	78±7.8	81±11	79±8.4	78±5.6	0.781
Duration of operation	248±42.6	245±39.8	249±53.4	246±49.6	0.821
Cross-clamping time	73±21.2	64±21.9	79±17.5	75±28.9	0.861
CPB time (min)	95±23.8	93±28.5	94±25.8	96±22.4	0.921
Gender n (%)					
Male	18 (78.1%)	21 (81.2%)	22 (85.1%)	18(78.2%)	p 0.918
Female	7 (25.1%)	6 (21.1%)	5 (17.1%)	7(25.1%)	

RESULTS

Table-I summarizes the demographic features of the individuals. In related to demographic features, there have been no differences between the two groups. Contempt a postoperative decrease in urine creatinine in altogether sets, the decrease was statistically substantial in Set II (p 0.06). (Table-II).

When compared to the other teams, individuals in Clusters 1 and 4 experienced the substantial rise (p 0.02) in urine microalbumin postoperatively. There seemed to be a substantial difference within different perspectives in Group II (p 0.06), there was really no substantial variance in Sets II and III (Table-II) (Fig.1).

A significantly massive rise in urine microalbumin proportion remained seen postoperatively in Sets 1, 2, 4 (p 0.02), and III

(p 0.06), despite not any variance among sets (Table-II) (Fig.2).

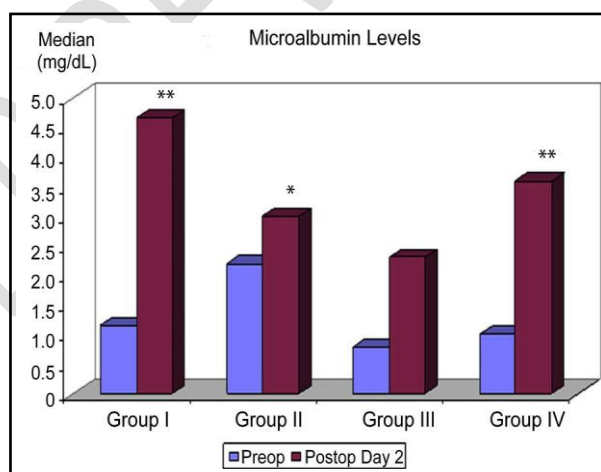


Fig.1: Level of microalbumin

Table-II: postoperative decrease in urine creatinine in altogether sets

		Group I	Group II	Group III	Group IV	p
		Mean±S	Mean±S	Mean±S	Mean±S	
Urine creatinine (mg/dL)	Preop	133.81±76.40	163.22±81.34	154.12±73.05	149.16±100.45	0.654
	Postop Day	124.96±80.74	117.23±78.21	123.25±150.84	123.80±77.11	0.993
Cystatin-C (mg/dL)	Preop	0.78±0.16	0.79±0.13	0.76±0.12	0.86±0.14	0.082
	Postop Day	0.93±0.20	0.93±0.22	0.85±0.12	0.92±0.22	0.396
Urine (mg/dL)	Preop	2.79±4.76	5.45±13.7	2.61±5.22	2.98±7.54	0.335
	Postop Day	9.29±18.44	5.67±6.33	2.73±2.19	5.35±7.70	0.688

Urine creatinin ratio (mg/dL)	◆P		0.002**	0.031*	0.264	0.005**	
	Preop		0.01±0.02	0.03±0.11	0.01±0.02	0.05±0.23	0.242
	Postop	Day	0.07±0.11	0.05±0.05(0.0	0.03±0.04	0.06±0.13	0.203
	◆P		0.002**	0.009**	0.023*	0.002**	

UNDER PEER REVIEW

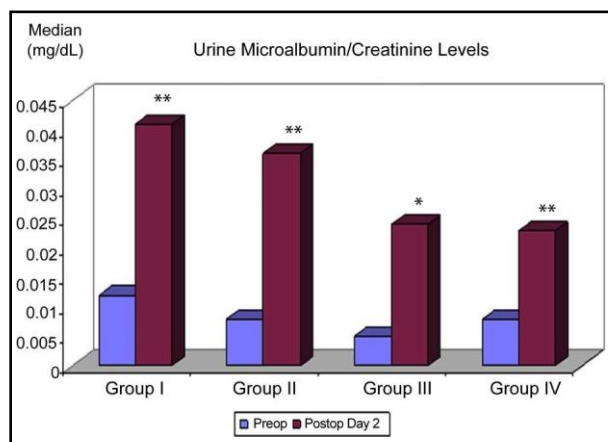


Fig.2: Urine micro albumin/creatinine levels

There would be a substantial rise in cystatin-c postoperatively in Sets 1, 2, and 3 (p 0.02), but not any substantial rise in Set IV (Table II) (Fig.3). In terms of necessity for extra diuretic usage, the research groups were comparable.

DISCUSSION

The pathogenesis of renal failure following open heart surgery is multifactorial and is commonly described by perioperative decreased cardiac output or the impact of CPB [6].

Cross-clamp time, length of CPB surpassing 72 minutes in specifically, pulsatile vs. non-pulsatile bypass flow, normothermic vs. hypothermic bypass, also bypass operation through or deprived of pump are other particular bypass-related possible causes commonly alluded to in the literature [7].

Non-pulsatile flow, renal hypoperfusion, and hypothermia were all linked to a decline in renal function.

Numerous recent studies, though, imply an elevated danger of renal impairment owing to hemodilution having hematocrit values under 26%. The research groups did not differ significantly in rappings of hemodynamic parameters, blood glucose, hemoglobin,

hematocrit stages (minimum Hct of 26), and hourly urine production in our group, including during surgery and standard follow-up [8].

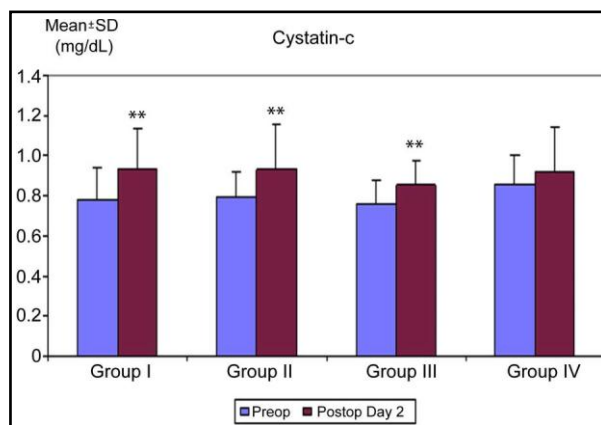


Fig.3:

Fig. 3. Cystatin-C level

There is some debate in the literature on the preventative benefits of dopamine also mannitol in CPB-associated kidney damage whether examined using alternative dosing schemes, timing, or routes. Such research, though, are often problematic due to limited sample numbers and discrepancies in variables employed to measure renal functioning. As a result, we sought to comparison properties of various mannitol and dopamine dosages also methods of administration using easily found laboratory measures [9].

Except for Group II (p 0.038), here remained not any substantial variances in urine creatinine levels when comparing between and within groups. However, we discovered that Group III outperformed some other groups in the following three tests:

Post-operative micro albumin levels increased significantly in Groups 1, 4 (p 0.02), in addition 2 (p 0.06). Inside-set increases in urine microalbumin/creatinine ratio were significant in Sets I, II, IV (p0.02), and III (p0.06). However, the differences between groups I and IV were statistical significance (p0.02). Prior research, meanwhile, demonstrated no substantial advantage for one of these criteria over the other [10].

CONCLUSION:

Urine microalbumin proportion and cystatin-c were found to give a past and stronger extrapolative

worth for identification of diabetic nephropathy than 24-hour urine microalbumin level and to serve as the criterion in pediatric RIFLE organization. Spite of substantial rise in urine microalbumin/creatinine ratio in T2 Set ($p < 0.02$), rise in cystatin-c in the current sample remained not statistically relevant ($p > 0.06$), indicating that somehow the current proportion may be an extra sensitive indicator for renal damage.

REFERENCES:

1. Hazle MA, Gajarski RJ, Aiyagari R, Yu S, Abraham A, Donohue J et al (2019) Urinary biomarkers and renal near-infrared spectroscopy predict intensive care unit outcomes after cardiac surgery in infants younger than 6 months of age. *J Thorac Cardiovasc Surg* 146(4):861-867.e1
2. Rosner MH, Okusa MD (2016) Acute kidney injury associated with cardiac surgery. *Clin J Am Soc Nephrol* 1(1):19–32
3. Torre S, Biondani E, Menon T, Marchi D, Franzoi M, Ferrarini D et al (2019) Continuous metabolic monitoring in infant cardiac surgery: toward an individualized cardiopulmonary bypass strategy. *Artif Organs* 40(1):65–72
4. Khwaja A (2019) KDIGO clinical practice guidelines for acute kidney injury. *Nephron Clin Pract* 120(4):c179-184
5. Jenkins KJ, Gauvreau K, Newburger JW, Spray TL, Moller JH, Iezzoni LI (2012) Consensus-based method for risk adjustment for surgery for congenital heart disease. *J Thorac Cardiovasc Surg* 123(1):110–118
6. Hoffman GM, Ghanayem NS, Tweddell JS (2020) Noninvasive assessment of cardiac output. *Semin Thorac Cardiovasc Surg Pediatr Card Surg Annu* 8(1):12–21
7. Ersoy C, Özyüksel A, Alkan Bozkaya T, Karaaslan P, Örmeci T, Ündar A et al (2016) Are perioperative near-infrared spectroscopy values correlated with clinical and biochemical parameters in cyanotic and acyanotic infants following corrective cardiac surgery? *Perfusion* 31(2):125–1
8. Ruf B, Bonelli V, Balling G, Hörer J, Nagdyman N, Braun S et al (2019) Intraoperative renal near-infrared spectroscopy indicates developing acute kidney injury in infants undergoing cardiac surgery with cardiopulmonary bypass: a case–control study. *Crit Care* 19(1):27
9. Choi D-K, Kim W-J, Chin J-H, Lee E-H, Don Hahm K, Yeon Sim J et al (2019) Intraoperative renal regional oxygen desaturation can be a predictor for acute kidney injury after cardiac surgery. *J Cardiothorac Vasc Anesth* 28(3):564–571
10. Owens GE, King K, Gurney JG, Charpie JR (2020) Low renal oximetry correlates with acute kidney injury after infant cardiac surgery. *Pediatr Cardiol* 32(2):183–188