Comparison of Cardioprotective Effects of Sevoflurane versus Propofol in Patients of Aneurysmal Subarachnoid Haemorrhage - Single Blind Randomized Clinical Study

Supriya[1], Deep Shikha[2], Madhulika Dubey[3]

Assistant Professor, Department of Anaesthesiology, Sarojini Naidu Medical College, Agra
Phone Number: 9719700248
Email: *myselfsupriya[at]gmail.com*

Assistant Professor, Department of General Surgery, Sarojini Naidu Medical College, Agra
Phone Number: 7870579888
Corresponding Author Email: *myselfdrdeepshikha[at]gmail.com*

Assistant Professor, Department of Intensive and Critical Care, Sarojini Naidu Medical College, Agra
Phone Number: 9899942628
Email: *madhulika16sep[at]gmail.com*

Abstract: *Background: Volatile anaesthetic agents have shown myocardial protective effects on heart and its use may show long term cardio - protective effects in immediate postoperative period. Therefore, this study aims to compare the cardio - protective role of sevoflurane MAC - 2 and sevoflurane MAC - 2.5 versus propofol during aneurysm clipping surgery in patients of subarachnoid haemorrhage SAH. Methods: A total of 90 patients aged 18 - 60 years undergoing clipping for aneurysmal SAH were included in the study. The patients were randomized using sealed - envelope technique into three groups of 30 each. Group 1 - Propofol (1 - 1.5 mg/kg) intravenous, Group 2 & Group 3 - Patients inhaled Sevoflurane. In group - 2, MAC was maintained at 2 and in group 3 MAC was maintained at 2.5. Blood samples were collected from patient before induction of anaesthesia, after surgery, and at 48 hrs post - surgery to detect cardiac injury markers troponin I, CK - MB and BNP levels. Continuous variables with normal distribution were compared between groups using one - way ANOVA, while continuous variables not normally distributed were compared using the Kruskal - Wallis test. The categorical data was analysed using Fischer - Exact test. P - value <0.05 was considered significant. Results: The cTnI level post - operative was found to be significantly higher in group 1 (0.29 ± 0.38 ng/ml) than in group 2 (0.02 ± 0.02 ng/ml) and group 3 (0.01 ± 0.003 ng/ml), p=0.0001. The CK - MB level increased significantly post - operative in group 1 (9.16 ± 13.93 ng/ml) than in group 2 (1.78 ± 1.64 ng/ml) and group 3 (1.69 ± 0.99 ng/ml), p=0.0001. The BNP level increased post - operative and at 48 hours post - operation in the three groups, however increase in BNP level was significantly higher in group 1 (134.53 ± 64.91 pg/ml & 257.88 ± 198.28 pg/ml, respectively) than group 3 (97.55 ± 74.58 pg/ml & 170.36 ± 173.52 pg/ml, respectively), p=0.011 & p=0.010, respectively. The significance of this study lies in its potential to inform clinical decisions regarding the use of anaesthetic agents in aneurysmal subarachnoid hemorrhage surgeries, which could improve patient outcomes by reducing cardiac injury. Conclusions: There is an increase in cardiac enzyme markers levels post - operatively but not raised to values that come under myocardial ischemia. Sevoflurane has a better cardio - protective profile than propofol in patients with SAH.*

Keywords: Sevoflurane, Propofol, Cardioprotective, Subarachnoid Hemorrhage, Aneurysm Clipping

Key Points:

- **Question:** Does Sevoflurane (MAC 2) and (MAC 2.5) are more cardio - protective than propofol in patients of aneurysmal subarachnoid haemorrhage. Is there any benefit of using Sevoflurane (MAC 2.5) over (MAC 2).
- **Findings:** Sevoflurane has better cardio - protective profile than propofol in patients with SAH. No addition benefit was observed for Sevoflurane (MAC 2.5).
- **Meaning:** Use of volatile anaesthetic agent sevoflurane reduces the cardiac injury and can be used during aneurysm clipping surgery in patients of subarachnoid haemorrhage.

Glossary of Terms:

ANOVA – Analysis of Variance
ASA - American Society of Anesthesiologists
BIS – Bispectral index
BMI – Body mass index
BNP – B - type natriuretic peptide
CK - MB – Creatine kinase – myocardial band
CONSORT - Consolidated Standards of Reporting Trials
CTRI - Clinical Trial Registry of India
cTnI – Cardio Troponin I
EF – Ejection fraction
ECG – Eelectrocardiogram
ICU – Intensive care unit
IPPV - Intermittent positive pressure ventilation
IV – Intravenous
LVEDP – Left ventricle end - diastolic pressure
MAC – Minimum alveolar concentration
NPO –Nill per oral
PO – Per oral
PONV – Post - operative Nausea and vomiting
SAH - Subarachnoid haemorrhage
SPSS - Statistical Package for Social Sciences

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1. Introduction

Subarachnoid haemorrhage SAH has an incidence of 6 to 7.5 cases per 100, 000 persons per year, accounting for approximately 6 to 10 of all the cerebrovascular accidents and is responsible for 22 to 25 of all cerebrovascular mortalities [3].[1] There are number of factors that lead to increased mortality and morbidity in patients of aneurysmal SAH and cardiac dysfunction is one of them [4].[2] Cardiac abnormalities in patients with SAH are associated with electrocardiographic changes, raised cardiac enzyme markers of myocardial ischemia in blood, and echocardiographic evidence of left ventricular dysfunction [5 - 8].[3-6]

Patients with SAH are often taken up for surgery in emergent condition, where there is very little time for the management of cardiac dysfunction. Thus anaesthetizing these patients is highly demanding in terms of fluctuating haemodynamic response and unpredictable response towards anaesthetic drugs. The cardiac depressant effects of anaesthetic agent can reduce the metabolic demand of cardiac function to some extent but the low cardiac output and arterial pressure might have deleterious effects on body organ perfusion and predisposes it to ischemic injury.

The modern volatile anaesthetic agents like sevoflurane and propofol have shown myocardial protective effects by virtue of their preconditioning in human studies [9 - 13].[7-10] These inhalational anaesthetics by virtue of having cardiac remodelling effects on heart during use may also show long term cardioprotective effects in immediate postoperative period. Therefore, the present study aims to compare the haemodynamics and cardioprotective role of sevoflurane (MAC 2) and sevoflurane (MAC 2.5) versus propofol during aneurysm clipping surgery in the patients of SAH.

2. Methods

The present study is a single blinded randomized comparative clinical study. A total of 90 patients from either sex with American Society of Anesthesiologists (ASA) grade I to III, Hess and Hunt grade 1 to 4, age between 18 to 60 years undergoing craniotomy and clipping for aneurysmal SAH were included in the study. The study was conducted in the department of Anestheology, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow, from month/year to month/year after obtaining approval from Institutional Ethical Committee. The patients were recruited after taking signed written informed consent. The study is registered in the clinical trial registry of India (CTRI/2015/01/005396). The manuscript adheres to the applicable Consolidated Standards of Reporting Trials (CONSORT) guidelines.

The patients with under 18 or over 60 years, ASA Grade IV or V, patients with severely impaired left ventricular function (EF < 30%, LVEDP > 18), with renal or liver impairment, patients with history of recent myocardial infarction (within 6 weeks), cardiac surgery (within previous 3 months), or repeated coronary surgery, patients having associated heart block and patients who refused to provide written informed consent were excluded from the study.

After enrolment, the severity of subarachnoid haemorrhage was graded clinically using the Hunt and Hess classification.

The patients were randomly divided into three groups of 30 each using sealed envelope technique for randomization. The research assistant involved in patient care confirmed patient eligibility and after obtaining written consent gave the sealed white envelope containing patient allocation and instruction for the anaesthesia technique to be used during the procedure to the attending anaesthesia resident.

Group 1 (Propofol group) (n = 30): Patients was induced (1 - 1.5 mg/kg) intravenous titrated to loss of verbal reflex and then anaesthesia was maintained by propofol infusion titrated to maintain BIS= 40 to 50.

Group 2 (Sevoflurane MAC 2 group) (n = 30): Patient was asked to inhale Sevoflurane (5%) in oxygen (3 l/min) and air (3 l/min) (50%) by facemask until loss of the eyelash reflex and MAC of sevoflurane was kept constant at 2.

Group 3 (Sevoflurane MAC 2.5 group) (n = 30): Patient was asked to inhale Sevoflurane (5%) in oxygen (3 l/min) and air (3 l/min) (50%) by facemask until loss of the eyelash reflex and MAC of sevoflurane was kept constant at 2.5.

A standard anaesthetic technique was used for all the patients. Patients were kept NPO 8 hours for solids and 2 hours for clear fluids before surgery.

Premedication: All patients were given premedication lorazepam 1.0 mg (PO) and ranitidine 150 mg (PO) was given night before surgery. Tab ranitidine 150 mg (PO) with a sip of water was repeated 2 hours before being shifted to OT. Midazolam 0.05 mg/kg IV was given in the OT before induction of anesthesia. If the patient was on any drug chronically, it was continued on the day of surgery.

Induction: In all the groups, endotracheal intubation was facilitated with vecuronium bromide 0.1 mg/kg. Following intubation patients were connected to ventilator for ventilation by volume-controlled mode with 50: 50% mixture of air and oxygen via closed circuit. Tidal volume was set at 8 - 10 ml/kg of body weight and respiratory rate of 10 - 12 breaths/minute. Minute ventilation was adjusted to maintain $EtCO_2$ of 35 ± 5 mm of Hg. Routine anaesthesia protocols were followed for the use of fentanyl (2 to 5 mcg/kg) for the maintenance of analgesia. All three groups received intermittent bolus of fentanyl to supplement the anaesthesia. Neuromuscular block was supplemented with intermittent bolus doses of vecuronium 0.02 mg/kg. The target levels of Sevoflurane was maintained throughout surgery and in the event of hypotension, noradrenaline infusion was started to titrate mean arterial pressure >70 mm of Hg.

At the end of surgery, residual muscle paralysis was reversed with neostigmine 0.05 mg/kg IV and glycopyrrolate 10 mcg/kg IV and tracheal extubation was performed once clinical criteria and train of four criteria were achieved. Patients with Hess and Hunt grade 3 or above, patients with haemodynamic unstability or patients with prolonged surgery and brain handling were not extubated in the operating room and shifted on mechanical ventilator support.

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All patients received dexamethasone 4 mg IV after induction of anaesthesia and ondansetron 4 mg IV at end of surgery, for post - operative nausea and vomiting (PONV) prophylaxis. Acetaminophen 15 mg/kg IV was infused over 15 minutes before start of surgery and then every 6 hours for 48 hours.

Measurements: Heart rate, systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP), central venous pressure (CVP) all these vitals were recorded at pre - induction, then hourly after post intubation till the end of surgery. ECG, ST segment analysis, inspired and expired concentration of sevoflurane, oxygen saturation, nasopharyngeal temperature, end tidal CO_2 concentration were monitored continuously. Arterial blood gas analysis was done pre - induction, post - induction and after surgery.

Blood samples were collected from the patient before induction of anaesthesia, at the end of surgery and at 48 hrs after surgery to detect the cardiac injury markers (troponin I, CK - MB and BNP) levels. The cardiac enzymes were measured using "Alere triage cardio3 panel" which is a fluorescence immunoassay for rapid quantitative determination of cTnI, CK - MB and BNP levels. The analytical sensitivity of the tests were 95th percentile of the measured results. Analytic sensitivity for each assay was cTnI <0.01 ng/ml, CK - MB<1.0 ng/ml and BNP<5 pg/ml. Measurable range for each assay was cTnI: 0.01 - 10 ng/ml, CK - MB: 1.0 - 80 ng/ml and BNP: 5 - 5, 000 pg/ml. In the present study Trop I >0.02 ng/ml, CK - MB > 4.3 ng/ml and BNP >149 pg/ml were considered as positive values.

All patients were kept in neuro surgery ICU for further observation and management until they were stable to be shifted to the ward. Early ambulation was done, if feasible. Patients were discharged from the ward when they were stable and needed no further observation.

Statistical analysis:
The data was entered into Statistical Package for Social Sciences (SPSS version 17, Boston, USA). The continuous variables which were normally distributed were compared between the three groups were analysed by using one - way ANOVA. The continuous variables which were not normally distributed were compared between the three groups were analysed by using non - parametric Kruskal - Wallis test, followed by post - hoc test. The categorical data was analysed by using Fischer Exact test. P value <0.05 was considered as statistically significant.

3. Results

The details of patients screened for enrolment, recruited and analysed is shown in figure 1. The mean ± standard deviation of age, weight, and BMI were found to be comparable between the 3 groups of the patients who underwent craniotomy and clipping for SAH (p=0.336, 0.628, 0.201, respectively) (Table 1). The ratio of male patients: female patients were comparable between group 1, group 2 and group 3 (8: 22, 6: 24 and 10: 20 respectively); p=0.510.

The cardiac injury marker cTnI level pre - operative was found to be 0.01 ng/ml in three groups, however the cTnI level increased significantly post - operative in group 1 (0.29 ± 0.38

ng/ml). The difference in the cTnI level post - operative between the groups was found to be significantly higher in group 1 than in group 2 (0.02 ± 0.02 ng/ml) and group 3 (0.01 ± 0.003 ng/ml), p=0.0001. The comparison of the cTnI between the groups is shown in Table 2. The raised cTnI level reduced to normal at 48 hours post - operation and no difference in the levels was found between the groups (p=0.303). Nineteen (63.3%) patients showed raised level of cTnI in group 1 post - operative as compared to 2 (6.7%) patients in group 2 and none in group 3 (p=0.0001). The distribution of normal and abnormal cTnI levels post - operation and 48 post - operation is shown in Table 3.

The second cardiac injury marker CK - MB level pre - operative was found to be comparable between 3 groups, 0.93 ± 0.25 ng/ml in group 1, 1.06 ± 0.35 ng/ml in group 2 and 1.03 ± 0.15 ng/ml in group 3 (p=0.753). The CK - MB level increased significantly post - operative in group 1 (9.16 ± 13.93 ng/ml) than in group 2 (1.78 ± 1.64 ng/ml) and group 3 (1.69 ± 0.99 ng/ml), p=0.0001. The comparison of the CK - MB between the groups is shown in Table 2. The raised CK - MB level reduced to normal at 48 hours post - operation and no difference in the levels was found between the groups (p=0.639). Twenty - two (73.3%) patients showed raised level of CK - MB in group 1 post - operative as compared to 3 (10%) patients in group 2 and 1 (3.3%) in group 3 (p=0.0001). The distribution of normal and abnormal CK - MB levels post - operation and 48 post - operation is shown in Table 3.

The third cardiac injury marker BNP level pre - operative was found to be comparable between 3 groups, 30.32 ± 24.96 pg/ml in group 1, 31.13 ± 42.43 pg/ml in group 2 and 21.20 ± 14.72 pg/ml in group 3 (p=0.342). The BNP level increased post - operative and 48 hours post - operation in all the 3 groups, however the increase in level of BNP was significantly higher in group 1 (134.53±64.91 pg/ml & 257.88±198.28 pg/ml, respectively) than group 3 (97.55±74.58 pg/ml & 170.36±173.52 pg/ml, respectively), p=0.011 & p=0.010, respectively (Table 2). Twenty three (76.7%) patients showed raised level of BNP in group 1 post - operative as compared to 15 (50%) patients in group 2 and 11 (36.7%) in group 3 (p=0.007). The raised BNP levels were observed in patients at 48 hours post - operation, 29 (96.7%) patients in group 1, 22 (73.3%) in group 2 and 21 (70%) in group 3 (p=0.02). The distribution of normal and abnormal BNP levels post - operation and 48 post - operation is shown in Table 3.

4. Discussion

In most of the cases of subarachnoid hemorrhage surgical intervention is required and this results in significant morbidity. There are number of factors that lead to increased mortality and morbidity in patients of aneurysmal SAH and cardiac dysfunction is one of them.[2]

The three study groups had equal number of patients (n=30), the patient characteristics in terms of age, weight, and BMI were comparable. All patients under anesthesia managed to attain normalization of hemodynamic state, so they did not showed significant difference in terms heart rate, systolic, diastolic and mean arterial blood pressure.

In the present study, the pre - operative blood samples of SAH patients showed normal levels of the cardiac enzyme markers (cTnI, CK - MB and BNP) while other studies reported increased levels in cardiac markers after SAH.[1, 11, 12] This might be due to difference in timing of sample collection. In the other studies first blood sample was taken on the time of hospital admission after the event while in present study the first sample was taken in pre - operating room after stabilization of patients and significant time elapsed since the event of SAH. It probably provided a window time for the cardiac enzyme levels to return to their near normal levels.

CK - MB levels have been used as cardiac injury biomarker in blood however it may also be released due to non - cardiac muscle injury and is not considered a reliable marker to quantify cardiac injury.[13, 14] Now–a - days, cardiac troponin I (cTnI) regulatory protein specific for cardiac muscle injury is used as the marker.[15] The cTnI may be released due to sub - lethal injury to cardiac muscles but the rise in level is less as compared to amount released after irreversible myocardial cell death and damage of contractile apparatus. So both sub - lethal and irreversible cardiac injury can be detected by measuring the rise in levels of cTnI.[13]

In this study, a significant rise in cTnI level was observed in group 1 (Propofol group) than the group 2 (Sevoflurane MAC 2) or group 3 (Sevoflurane MAC 2.5) (p=0.0001). No significant difference was found between group 2 and group 3 (p=0.94). The cTnI levels did not showed significant difference at 48 hours post - operation (p>0.05). Similarly, a significant rise in CK - MB level was observed in group 1 (Propofol group) than the group 2 (Sevoflurane MAC 2) or group 3 (Sevoflurane MAC 2.5) (p=0.001). No significant difference was found between group 2 and group 3 (p=0.94). The CK - MB levels did not show significant difference at 48 hours post - operation (p>0.05). Bharti et al., (2008) studied cardio protective role of sevoflurane in coronary artery bypass grafting surgery and reported that the sevoflurane had better cardio - protective profile in terms lesser degree of rise in cardiac enzyme markers in patients who were anaesthetized with sevoflurane than the propofol.[16]

Yildirim et al., (2009) compared cardioprotective effects of sevoflurane, isoflurane and propofol on patients posted for coronary surgery and concluded that inhalational anesthetic as sevoflurane preserved cardiac functions better in comparison with propofol and patients had less evidence of cardiac injury with the inhalational agents than propofol.[17]

Bassuoni et al., (2012) conducted a study on the patients with coronary artery disease undergoing vascular surgery and found that patients receiving sevoflurane as anesthetic technique had significantly lower rise of cTnI at 6 hrs post - operatively and effect last for 48 hrs than patients receiving propofol for the same procedure. Moreover, there was also significant decrease in ischemic events, ECG changes and overall duration of hospital stay in patients receiving sevoflurane.[18] The patients in present study did not figure out coronary artery disease, as cTnI levels did not increase enough to call it myocardial ischemia (rise is < 5 fold of normal value). However, the elevated levels suggest an element of cardiac stress which was not to the extent to be categorized as myocardial ischemia.

Along with cTnI and CK - MB, a BNP level at the same point of times was measured to assess its utility for the heart failure. Normal BNP levels rule out heart failure in emergency setting, although in lack of the specificity for cardiac origin its elevated levels may not confirm the heart failure.[19] In the study increased BNP levels was observed post - operatively and the level continued to rise when measured at 48 hrs. Rise in BNP level was more in group 1 patients and no significant difference was found between group 2 and 3. Thus it distinctly affirmed an element of acute cardiac failure even after surgery in SAH patient in postoperative period although ischemic element got corrected. Since elevated levels in cardiac enzymes was less pronounced in patients receiving sevoflurane anesthesia versus propofol anesthesia, it suggests better cardio - protective profile of the sevoflurane.

Maintaining twice the MAC level of sevoflurane is found to have cardio - protective effects like sevoflurane levels to MAC - 2.5, and sevoflurane MAC 2.5 did not add any further protective effects in terms of changes in cardiac injury enzyme levels.

5. Conclusion

The present study found that there is some amount of cardiac stress present in patients immediately in post - operative period. There is an increase in cardiac enzyme markers post - operatively but they are not raised to the values that come under myocardial ischemia. So, there must be some cardiac insult during the operative procedure other than ischemia. Sevoflurane has a better cardio - protective profile than propofol in patients with SAH.

6. Limitations

Small sample size is the main limitation of the study. So further study needed for wide application of the study.

Author Contribution
Supriya – The author helped in study planning, execution, analysis and writing of manuscript.
Deep Shikha – The author helped in analysis and writing of manuscript.
Madhulika Dubey
Designation: Assistant Professor
Department: Intensive and Critical Care
Phone Number: 9899942628
E - mail Address: madhulika16sep[at]gmail.com

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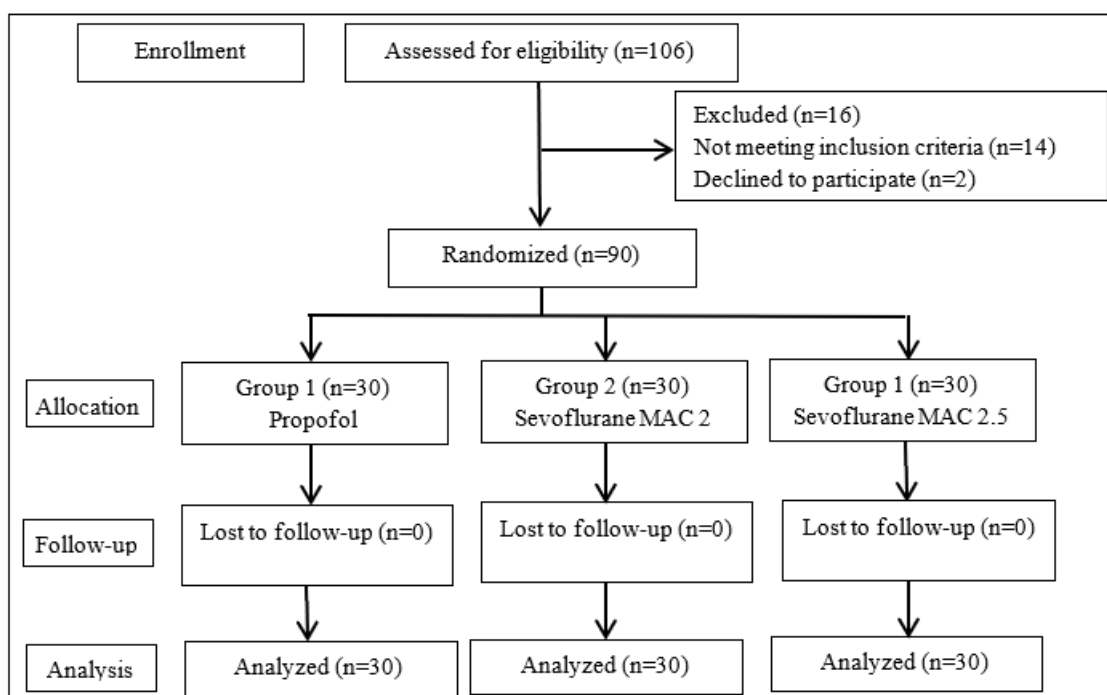
Figure 1: COHORT Flow Chart

Table 1: Baseline demographic profile of the study groups

Characteristics	Group 1 (n=30)	Group 2 (n=30)	Group 3 (n=30)	p - value
Age (Years)	50.40 ± 9.19	51.93 ± 9.53	48.27 ± 10.05	0.336
Weight (Kg)	59.97 ± 8.56	60.20 ± 8.74	61.97 ± 8.98	0.628
BMI (Kg/m^2)	22.65 ± 3.22	23.25 ± 2.98	24.14 ± 3.42	0.201

Table 2: Cardiac injury biomarker profile of the study groups

Cardiac injury biomarkers	Group 1 (n=30)	Group 2 (n=30)	Group 3 (n=30)	p - value		
	1	2	3	1vs2	1vs3	2vs3
cTnI (ng/ml)						
Pre - operative	0.01±0.00	0.01±0.00	0.01±0.00	-	-	-
Post - operative	0.29±0.38	0.02±0.02	0.01±0.003	0.0001*	0.0001*	0.94
At 48 hrs	0.02±0.03	0.03±0.11	0.01±0.00	0.401	0.124	0.509
CK - MB (ng/ml)						
Pre - operative	0.93±0.25	1.06±0.35	1.03±0.15	0.522	0.522	1.000
Post - operative	9.16±13.93	1.78±1.64	1.69±0.99	0.001*	0.0001*	0.96
At 48 hrs	1.91±1.86	1.27±0.67	1.43±1.48	0.418	0.418	0.976
BNP (pg/ml)						
Pre - operative	30.32±24.96	31.13±42.43	21.20±14.72	0.238	0.204	0.667
Post - operative	134.53±64.91	124.47±99.80	97.55±74.58	0.230	0.011*	0.327
At 48 hrs	257.88±198.28	207.47±187.99	170.36±173.52	0.208	0.010*	0.177

Table 3: Distribution of abnormal cardiac injury biomarker level post - operation and at 48 post - operation in the study groups

Cardiac injury biomarkers	Group 1 (n=30)	Group 2 (n=30)	Group 3 (n=30)	p - value		
	1	2	3	1vs2	1vs3	2vs3
cTnI (ng/ml)						
Post - operative						
Normal	11 (36.7%)	28 (93.3%)	30 (100%)	0.0001*	0.0001*	0.491
Abnormal	19 (63.3%)	2 (6.7%)	0 (0%)			
At 48 hrs						
Normal	26 (86.7%)	28 (93.3%)	30 (100%)	0.671	0.112	0.491
Abnormal	4 (13.3%)	2 (6.7%)	0 (0%)			
CK - MB (ng/ml)						
Post - operative						
Normal	8 (26.7%)	27 (90%)	29 (96.7%)	0.0001*	0.0001*	0.96
Abnormal	22 (73.3%)	3 (10%)	1 (3.3%)			
At 48 hrs						
Normal	26 (86.7%)	30 (100%)	29 (96.7%)	0.418	0.418	0.612
Abnormal	4 (13.3%)	0 (0%)	1 (3.3%)			
BNP (pg/ml)						
Post - operative						
Normal	7 (23.3%)	15 (50%)	19 (63.3%)	0.060	0.004*	0.435
Abnormal	23 (76.7%)	15 (50%)	11 (36.7%)			
At 48 hrs						
Normal	1 (3.3%)	8 (26.7%)	9 (30%)	0.026*	0.012*	1.000
Abnormal	29 (96.7%)	22 (73.3%)	21 (70%)			

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