

# CAD patients who were undergoing elective surgical procedure under general anesthesia: Clinical correlates

<sup>1</sup>Dr. Vishal Mallappa Kappattanavar, <sup>2</sup>Dr. PG Raghavendra, <sup>3</sup>Dr. Chandana MH, <sup>4</sup>Dr. Jyoti Magar

<sup>1</sup>Senior Resident, Department of Anesthesia, GIMS, Gadag, Karnataka, India

<sup>2</sup>Assistant Professor, Department of Anesthesia, RIMS, Raichur, Karnataka, India

<sup>3</sup>Assistant Professor, Department of Anesthesia, VIMS, Ballari, Karnataka, India

<sup>4</sup>Associate Professor, Department of Anesthesia, Lokmanya Tilak municipal medical college and general hospital, Mumbai, India

## Corresponding Author:

Dr. Jyoti Magar

## Abstract

CAD and stroke are the predominant causes and are responsible for >80% of CVD deaths. The Global Burden of Disease study estimate of age-standardized CVD death rate of 272 per 100000 populations in India is higher than the global average of 235 per 100000 populations. In India of particular concern is its accelerated buildup, the early age of disease onset in the population and the high case fatality rate. After getting approval from institutional ethics committee, study was carried out in all patients fulfilling the inclusion and exclusion criteria during the study period. Patient diagnosed or known case of CAD either from history, investigations were included in the study after obtaining written valid informed consent. 2D Echo of patient diagnosed or known CAD was routinely done in our institution mainly to know the ventricular function. The number of patients having DM was comparable in the two groups (p value-0.644). The percentage of DM patients in group A and group B being 40% and 35% respectively. The time taken for intubation in two groups was comparable, with the mean duration being 19.5s in group A and 19.4s in group.

**Keywords:** CAD patients, Clinical correlates, DM

## Introduction

Coronary Artery Disease (CAD) is the leading cause of both mortality and forgone healthy years of life among working-age adults (15-69 years) in South Asia including India. A quarter of all mortality is attributable to CVD. CAD and stroke are the predominant causes and are responsible for >80% of CVD deaths. The Global Burden of Disease study estimate of age-standardized CVD death rate of 272 per 100000 populations in India is higher than the global average of 235 per 100000 populations. In India of particular concern is its accelerated buildup, the early age of disease onset in the population and the high case fatality rate. Behavioral risk factors, including tobacco use, physical inactivity and unhealthy diet, are responsible for about 80% of CAD and cerebrovascular disease. The prevalence of IHD in 1960 in urban India was 2% and increased 7 fold to  $\approx$ 14% by 2013. The Macroeconomic Commission for Health estimated that the absolute number of CAD patients in India will increase from 36 million in 2005 to 62 million in 2015 (a  $\approx$ 70% increase) <sup>[1, 2]</sup>.

With increasing incidence of coronary artery disease, the number of patients with CAD with or without interventions coming for non-cardiac surgical procedures is also increasing. Also the comorbidities associated with CVD have seen a significant rise in the past decade. In India, the age-standardized annual stroke incidence rate is 154 per 100000 per year. Hypertensive heart disease, among other

CVDs, is a significant problem in India, with 261694 deaths in 2013; this is an increase of 138% in comparison with the number of deaths in 1990 [3, 4].

The administration of anaesthesia to the patients with preexisting cardiac disease is a challenge. These patients have increased risk of myocardial infarction (MI), conduction disturbances, morbidity and mortality during the perioperative period. Patients with CAD presents to the anaesthetist with varying degree of cardiac dysfunction. Patient may present with history of angina pectoris with coronary blood flow compromised either at rest or in response to anxiety or during exercises. They may give past history of one or more episodes of MI. Though the majorities of patients with significant coronary disease present for anaesthesia and surgery, but give neither a history nor symptoms which identify them. Coronary artery disease results in regional rather than global ischemic changes. It is important to recognize factors which result in redistribution of coronary blood flow, as much as those causing either a global decrease of coronary blood flow or an increase of myocardial oxygen uptake. Principle in providing safe anaesthesia for the patient with coronary artery disease is to avoid those conditions which precipitate myocardial ischemia, specifically to avoid tachycardia, with or without hypertension or hypotension. The changes of arterial pressure and heart rate which occur during and after anaesthesia reflect a balance between the direct haemodynamic effects of anaesthetic agents and other drugs, the reflex haemodynamic responses to such changes of arterial pressure, the reflex haemodynamic responses to the noxious stimuli associated with surgery [5, 6].

Induction of general anaesthesia is a critical period in these patients, especially in presence of LV dysfunction. The considerations during induction of anaesthesia include haemodynamic stability, attenuation of the stress responses to laryngoscopy and maintenance of balance between myocardial oxygen demand and supply. Laryngoscopy is considered to be the most potent noxious stimuli in respect of its effects on autonomic nervous activity. It may cause reflex activation of sympathetic nervous system which may precipitate episodes of myocardial ischemia during anaesthesia. These responses may occur even when anaesthetic concentrations are high enough to suppress sensory, motor and breathing responses [7, 8].

Premedication with opioids, beta adrenoreceptor blockers, infusion of nitroglycerin and administration of lignocaine have been found to be effective in blunting this response. The volatile anaesthetics were found to be less effective in blocking the heart rate and pressor responses to laryngoscopy and intubation. Also at higher concentrations volatile anaesthetics especially Isoflurane may cause myocardial ischemia and it may be prudent to avoid its use in circumstances where high concentrations may be necessary.

## Methodology

### Materials & Methods

- A. Design prospective/retrospective: Prospective observational study.
- B. Place of the study: Various Operation Theatres at tertiary health centre.
- C. Sample size: Study was conducted in 80 CAD patients who were undergoing elective surgical procedure under general Anaesthesia during the study period.
- D. Sampling method: Convenient consecutive consenting sample.

### Inclusion criteria

- a) All elective surgeries except cardiac surgery.
- b) Patients with CAD diagnosed with changes on ECG and known case of CAD with ejection fraction (EF) >10%.
- c) Age >40 & <80 years.
- d) American Society of Anesthesiology grading risk (ASA) II-III.
- e) Haemodynamically stable.
- f) Surgery duration >2hours.

## Exclusion criteria

- a) Patient's refusal for consent.
- b) History of allergy to etomidate.
- c) Predicted difficult airway.
- d) Intubation duration more than 30s.
- e) Congestive cardiac failure.
- f) Persistent Arrhythmias.
- g) Severe systemic non cardiac disease.
- h) Pregnant and lactating patients.

After getting approval from institutional ethics committee, study was carried out in all patients fulfilling the inclusion and exclusion criteria during the study period. Patient diagnosed or known case of CAD either from history, investigations were included in the study after obtaining written valid informed consent. 2D Echo of patient diagnosed or known CAD was routinely done in our institution mainly to know the ventricular function. CAD patients who were undergoing elective non cardiac surgery and who were given etomidate as an induction agent were observed. Thorough pre anaesthetic check-up was done by routine anaesthetist.

Routine and specific investigations were noted. Preoperative medications taken by the patient were noted. The patient then underwent general anaesthesia as per standard protocol as decided by the anaesthetist incharge. All patients were monitored with electrocardiogram, non-invasive blood pressure (NIBP), pulse oximeter and capnograph. Intra-arterial BP monitoring and central venous pressure monitoring if done were noted.

## Results

**Table 1:** Association between Study Group and Gender (N=80)

Gender	Group	
	EF <45(group A) (n=40) n (%)	EF >45(group B) (n=40) n (%)
Female	17 (42.5)	17 (42.5)
Male	23 (57.5)	23 (57.5)
Chi-Square Test, P Value = 1.000, Not Significant		

As per the above table the gender distribution was comparable between the groups (p value- 1.0) with the percentage of M: F being 57.5:42.5 in both groups.

**Table 2:** Association between Study Group and ASA (N=80)

ASA	Group	
	EF <45(group A) (n=40) n (%)	EF >45(group B) (n=40) n (%)
2	18 (45.0)	19 (47.5)
3	22 (55.0)	21 (52.5)
Chi-Square Test, P Value = 0.822, Not Significant		

The ASA grade of patients in 2 groups were similar (p value- 0.822), with each group having 45% of patients in ASA 2 group and 55% of patients in ASA 3.

**Table 3:** Association between Study Group and HTN (N=80)

	Group	
	EF <45(group A) (n=40) n (%)	EF >45(group B) (n=40) n (%)
Yes	20 (50.0)	21 (52.5)
No	20 (50.0)	19 (47.5)
Chi-Square Test, P Value = 0.823, Not Significant		

The percentages of patients with hypertension in 2 groups were similar (p value-0.823) with 50% of patients in group A and 52.5% of patients in group B having hypertension.

**Table 4:** Association between Study Group and DM (N=80)

	Group	
	EF <45(group A) (n=40) n (%)	EF >45(group B) (n=40) n (%)
Yes	16 (40.0)	14 (35.0)
No	24 (60.0)	26 (65.0)
Chi-Square Test, P Value = 0.644, Not Significant		

The number of patients having DM was comparable in the two groups (p value-0.644). The percentage of DM patients in group A and group B being 40% and 35% respectively.

**Table 5:** Association between Study Group and Stroke (N=80)

	Group	
	EF <45(group A) (n=40) n (%)	EF >45(group B) (n=40) n (%)
Yes	3 (7.5)	3 (7.5)
No	37 (92.5)	37 (92.5)
Chi-Square Test, P Value = 1.000, Not Significant		

The number of patients with history of stroke was comparable (p value-1.0), with 7.5% of patients having history of stroke in each group.

**Table 6:** Association between Study Group and NYHA Classification (N=80)

NYHA Classification	Group	
	EF <45(group A) (n=40) n (%)	EF >45(group B) (n=40) n (%)
1	3 (7.5)	3 (7.5)
2	22 (55.0)	21 (52.5)
3	13 (32.5)	14 (35.0)
4	2 (5.0)	2 (5.0)
Chi-Square Test, P Value = 0.996, Not Significant		

The NYHA classification of the patients in 2 groups was comparable (p value-0.996). In group A the percentage of patients in NYHA class I, II, III and IV were 7.5%, 55%, 32.5% and 5% respectively. In group B the percentage of patients in NYHA class I, II, III and IV were 7.5%, 52.5%, 35% and 5% respectively.

**Table 7:** Comparison of Duration of Intubation between 2 Study Groups (N=80)

Parameter	Group		P Value
	EF<45(group A) (n=40) Mean (SD)	EF >45(group B) (n=40) Mean (SD)	
Duration of Intubation	19.50 (2.97)	19.47 (2.97)	0.970
Unpaired t Test, P Value Not Significant			

The time taken for intubation in two groups was comparable, with the mean duration being 19.5s in group A and 19.4s in group.

## Discussion

80 cases were observed, 40 patients with LVEF<45% and 40 patients with LVEF>45%. The demographic profile was comparable between the two groups in our study. The mean age, the ratio of male and female patients was comparable between the 2 groups and the difference was statistically not significant.

The ASA grade of patients in the two groups was comparable and statistically not significant (p value-0.822). 47.5% and 52.5% patients belonged ASA 2 & ASA 3 respectively in patients with EF < 45%. 45% and 55% patients belonged to ASA 2 & ASA 3 in patients with EF>45%.

The prevalence of HTN and DM in both the groups were comparable, with 52.5% of patients in patients with EF<45% and 50% of patients in patients with EF > 45% having HTN. The difference was statistically insignificant (p value-0.823) and 35% of patients with EF < 45% and 40% of patients with EF > 45% were having DM (p value-0.644) Rachna *et al.* studied the association of risk factors in ischemic heart disease patients and observed that 62% of cases had HTN whereas only 20% of control had HTN and 50% of cases and 18% of control had DM, indicating that HTN & DM were a risk factor for IHD [9].

Hence in this study, the groups were comparable with respect to age ,sex ,Weight, BMI , associated comorbidities ,ASA grading. In both group, patients requiring > 30 sec for intubation were excluded from study as it can influence haemodynamic changes at intubation. Opioids are used in CAD to prevent intubation response as they do not cause myocardial depression and have very few cardiovascular side effect [10]. Fentanyl and midazolam also prevent myoclonus associated with etomidate induction. (38) In both group IV fentanyl was given in the dose 2 mcg /kg and IV midazolam given in the dose of 0.02 mg /kg 3 minutes prior to induction to prevent response to intubation and laryngoscopy which is our routine practice. So the difference in the haemodynamic could be attributed to the effect of etomidate on haemodynamic according to left ventricular function.

## Conclusion

The NYHA classification of the patients in 2 groups was comparable (p value-0.996). In group A the percentage of patients in NYHA class I, II, III and IV were 7.5%, 55%, 32.5% and 5% respectively. In group B the percentage of patients in NYHA class I, II, III and IV were 7.5%, 52.5%, 35% and 5% respectively.

## References

1. Aggarwal S, Goyal VK, Chaturvedi SK, Mathur V, Baj B, Kumar A. A comparative study between propofol and etomidate in patients under general anesthesia. Brazilian J Anesthesiol (English Ed [Internet]. 2016;66(3):237-41. Available from: <http://dx.doi.org/10.1016/j.bjane.2014.10.005>
2. Forman SA. Clinical and Molecular Pharmacology of Etomidate. NIH Public Access. 2011;114(3):695-707.

3. Stoelting. Stoelting's pharmacology and physiology in anaesthetic practice. 5th ed. Pamela, James S, editor. Philadelphia: Wolters Kluwer, 2016, 168-171.
4. Miller Ronald D. miller's anaesthesia. 8th ed. Neal, Lars, Lee J, editor. Philadelphia: Elsevier Saunders, 2015, 850-854.
5. Gooding JM, Weng JT, Smith RA, Berninger GT, Kirby RR. Cardiovascular and pulmonary responses following etomidate induction of anesthesia in patients with demonstrated cardiac disease. *Anesth Analg.* 1979;58(1):40-1.
6. Harris CE, Murray Jmarmgamm AM. Summary. Effects of thiopentone, etomidate and propofol on the haemodynamic response to tracheal intubation. 1988;43:32-6.
7. Larsen R, Rathgeber J, Bagdahn A, Lange H, Rieke H. Effects of propofol on cardiovascular dynamics and coronary blood flow in geriatric patients: A comparison with etomidate. *Anaesthesia.* 1988;43:25-31.
8. Boer F, Bovill JG, Ros P, Ommen H Van. Effect of thiopentone, etomidate and propofol on systemic vascular resistance during cardiopulmonary bypass. *Br J Anaesth.* 1991;67(1):69-72.
9. Reinhard Haessler, Christian Madler, Sven Klasing DS and KP. Propofol/Fentanyl Versus Etomidate/Fentanyl for the induction of anaesthesia in patients with aortic insufficiency and Coronary Artery Disease. *Journal of Cardiothorac Vasc.* 1992;6(2):173-80.
10. Cheong KF, Choy JML. Sevoflurane-fentanyl versus etomidate-fentanyl for anesthetic induction in coronary artery bypass graft surgery patients. *J Cardiothorac Vasc Anesth.* 2000;14(4):421-4.