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Change in Left Ventricular Peak Global Longitudinal Strain in Patients Undergoing Uneventful On-Pump Coronary Artery Bypass Grafting Surgery

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ABSTRACT

Objectives: Immediately after coronary artery bypass grafting (CABG) surgery, the left ventricular (LV) function measured by peak LV global longitudinal strain (GLS) is influenced by general anesthesia, positive pressure ventilation, myocardial protection technique used, the effect of myocardial revascularization, and the effect of inotropic and vasopressor agent used. Therefore, we intended to perform a study evaluating the change in the LV function, measured by peak LV GLS, from the immediate post-operative period to the immediate pre-hospital discharge period (7–10 days after surgery) in patients undergoing uneventful, on-pump CABG surgery and having an uneventful early post-operative course.

Material and Methods: In patients aged 30–65 years, having LV ejection fraction (LVEF) >50%, undergoing elective, multiple (>2) graft, on-pump CABG surgery by a single surgeon and having uneventful post-operative course, peak LV GLS was measured in the immediate post-operative period by TEE and at the time of hospital discharge (post-operative day 7–10) by transthoracic echocardiography. The association between the change in the peak LV GLS and the vasoactive inotropic score (VIS) in the immediate post-operative period was also checked.

Results: Analysis of data from 51 participants revealed a significant decline in peak LV GLS from $-12.8 \pm 3.8\%$ (mean ± standard deviation [SD]) in the immediate post-operative period to $-10.2\pm2.4\%$ (mean ± SD) in the immediate pre-discharge period (P = 0.000). Heart rate and cardiac index decreased significantly, while mean arterial pressure and systemic vascular resistance index increased significantly from the immediate post-operative period to the immediate pre-discharge period.

Conclusion: Low-risk patients with normal LVEF undergoing multivessel CABG surgery with uneventful intraoperative and early post-operative (during index hospital stay) course have a significant decline in peak LV GLS from the immediate intraoperative period. This decline is not significantly associated with either immediate pre-operative peak LV GLS or VIS in the immediate post-operative period.

Keywords: Coronary artery bypass, Global longitudinal strain, Post-operative period

INTRODUCTION

Systolic function of the left ventricle (LV) is used mainly to predict perioperative mortality and morbidity in patients undergoing coronary artery bypass grafting (CABG) surgery.^[1] Trans-esophageal echocardiography (TEE) plays the main role in assessing LV function intra-

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operatively in patients undergoing CABG surgery.^[2] While LV ejection fraction (EF), measured by Simpson's biplane method, is the most widely used parameter and is considered a cornerstone for measurement of LV systolic function as well as for prediction of outcome, it has various limitations such as inter and intra-observer variability, load dependency, and tethering.^[3,4] Two-dimensional (2D) speckle tracking imaging (STE) enables measurement of myocardial deformation, which provides high quality, precise, and objective information regarding regional and global myocardial function in real time. Global longitudinal strain (GLS) by STE can identify subtle LV dysfunction, has significantly less inter and intra-operator variability than EF, and is more precise method for evaluation of LV function with/without echocardiographic training.^[4,5] Systolic LV function assessed using GLS is particularly important as it can reveal systolic abnormalities despite normal LVEF, which are associated with less favorable outcome.^[6] LVEF is predominantly related to LV circumferential shortening, whereas GLS depicts LV longitudinal shortening. As the vulnerable subendocardial myofibers are responsible for the longitudinal shortening of LV, GLS is able to detect subtle changes in myocardial function.^[3,6]

During cardiac surgery, the LV function is measured intraoperatively by TEE. However, in the immediate post-CABG period, the LV function is influenced by general anesthesia, positive pressure ventilation, myocardial protection technique used, the effect of myocardial revascularization, and the effect of inotropic and vasopressor agent used.^[7] However, the magnitude of the combined effect of the above-mentioned confounding factors in the immediate post-operative period on LV function is not known. Moreover, although the change of peak LV GLS over the perioperative period of CABG surgery has been studied;^[8] to the best of our knowledge, literature is scarce specifically regarding the change in peak LV GLS from immediate postoperative period to immediate pre-hospital discharge period (Postoperative day 7-10), i.e., the change of peak LV GLS over the early postoperative period in patients undergoing CABG surgery. As the effects of anesthesia, positive pressure ventilation, cardioplegia, inotropes, and vasopressors wean off by the time of hospital discharge (postoperative day 7-10), finding the difference between peak LV GLS measured in the immediate post-operative period and that measured just before hospital discharge (postoperative day 7-10) can estimate the combined effect of all the intra-operative confounders on peak LV GLS.

Therefore, we intended to perform a study evaluating the change in the LV function, measured by peak LV GLS, from the immediate post-operative period to the immediate pre-hospital discharge period (7–10 days after surgery) in patients undergoing uneventful, on-pump CABG surgery

and having uneventful early post-operative (during hospital stay) course.

MATERIAL AND METHODS

Study design

This prospective, observational study was conducted between June 2022 and July 2023.

The study was commenced after the clearance of the ethics committee of the institute and trial registration at the Clinical Trial Registry of India (CTRI/2022/04/042217). Written, informed consent was taken from the patients before enrolling them into the study.

Inclusion criteria

Patients in the age group of 30–65 years of either gender, undergoing elective, isolated multiple grafts (>2 grafts) on-pump coronary artery bypass grafting surgery were included.

Exclusion criteria

Patients with any or combination of the following- declining to give consent, having abnormal (<50%) pre-operative LVEF, not in sinus rhythm at the time of performing TEE/transthoracic echocardiography (TTE), on pacemaker, recent (<28 days) acute coronary syndrome, having more than mild valvular regurgitation and/or stenosis, emergency or re-do CABG, having contraindications to insertion of TEE transducer, having low cardiac output syndrome/ mechanical cardiac support/prolonged (>24 h) mechanical ventilation/suspected or confirmed graft occlusion or kink/ re-exploration/respiratory or liver or kidney dysfunction/ significant arrhythmia/sepsis/shock/cardiac arrest in postoperative period, having poor TTE window at the time of hospital discharge, required inotropic/vasopressor support or mechanical cardiac support before induction of anesthesia were excluded.

Upon patient's arrival into operating room, standard monitors were attached and invasive arterial as well as central venous access were secured. Subsequently, general anesthesia was induced using balanced anesthesia technique, comprising fentanyl (5–10 mcg/kg), propofol (1–2 mg/ kg), and vecuronium (0.1 mg/kg). After endotracheal intubation, the patient was ventilated as directed by the anesthesia faculty posted in the operating room and anesthesia was maintained using inhalational isoflurane with a target BiSpectral Index (BIS) of 50. A 6VT-D TEE probe was inserted in all patients after induction of general anesthesia and insertion of endotracheal tube, using standard maneuvers. The surgery was performed using mild hypothermic (34°C) cardiopulmonary bypass (CPB). Intermittent cold (6–8°C) blood cardioplegia was used to accomplish myocardial protection, delivered both through antegrade and retrograde route. All the surgeries involved a single cardiothoracic and vascular surgical faculty with more than 5 years' experience. Milrinone (0.375–0.75 mcg/kg/min) was used as inotrope of choice when cardiac index (C.I) measured by TEE < 2.2 L/m² body surface area (BSA)/min after ensuring optimal LV filling (LV end-diastolic area [LV EDA] 8–14 cm²) at the end of CPB. Noradrenaline was used as vasopressor of choice when mean arterial pressure (MAP) < 65 mm Hg with C.I > 2.2 L/m²BSA/min with optimal LV filling post-CPB.

The study parameters of the time point immediate postoperative period were recorded using TEE immediately before shifting the patient from the operating room after the completion of the surgery, maintaining C.I > 2.2 L/ m² BSA/min, MAP > 65 mmHg, BIS 40–60 with isoflurane and maintaining mechanical ventilation with Positive end expiratory pressure (PEEP) 4–6 cm H₂O, respiratory rate 12– 15, Tidal volume (Vt) 6–8 mL/kg of ideal body weight. The TEE parameters were recorded by an anesthesia faculty (with more than 5 years of experience) different from the faculty posted in the operating room.

After shifting the patient from the operating room to the cardiac surgical intensive care unit, the patient was mechanically ventilated as per institutional protocol. Further hemodynamic and ventilatory management was done as per the anesthesia and cardiac surgical faculty involved for the patient. Liberation from mechanical ventilation was decided as per standard criteria. Weaning off vasopressorinotropic support was done as per the guidance of anesthesia and cardiac surgical faculty involved for the patient while ensuring C.I > 2.2 L/m² BSA/min (by echocardiography) and MAP > 65 mmHg, arterial lactate < 2 mmol/L. Removal of invasive lines, discharge from ICU, and initiation of post-operative medications, such as beta blocker, statin, and anti-platelet agents was guided by the involved cardiac surgical faculty. The patient was discharged from the hospital as determined by the cardiac surgical faculty. The study parameters of the timepoint immediate pre-hospital discharge were recorded using TTE just before discharge of patient from the hospital.

Before anesthesia induction and at the time of hospital discharge, cardiology faculty with more than 5 years' experience performed TTE with the patient in supine/left lateral position to measure peak LV GLS, LV EDA, IVC diameter (both inspiratory and expiratory), left ventricular outflow tract (LVOT) velocity time integral (VTI). Transthoracic echocardiographic was performed using a dedicated GE Vivid E9 workstation (GE Healthcare Vingmed Ultrasound AS, Horten, Norway) with M5S-D (1.5–4.5 MHz) transthoracic probe. The measurements taken before

induction of anesthesia (pre-induction) were taken as the baseline values of the same.

Peak LV GLS was estimated using video clips recorded at apical four chamber (A4C), two chamber (A2C) and three chamber (A3C) views with similar heart rate (HRs) (within ten beats/minute), as described by European Association of Cardiovascular Imaging/American Society of Echocardiography.^[9] Strain analysis was performed offline using automated function imaging of dedicated software (Echopac 113, GE) with the automatic delineation of endocardial and epicardial borders, tracking the speckles throughout the cardiac cycle, thus deriving the peak GLS. During TEE, peak LV GLS was obtained by acquiring mid esophageal 4 chambers (ME4C), 2 chambers (ME2C), and mid esophageal long axis (ME LAX) views and subsequently analyzed the video clips utilizing similar methodology as done during TTE. Normal value for peak LV GLS was taken as -17% as per Menting et al.[10] LVEF, LV EDA, LVOT diameter, LVOT VTI, and IVC diameter were measured as per methods described by ASE.[11]

HR, MAP, and central venous pressure (CVP) were recorded at the time of TTE before anesthesia induction and during TEE at the immediate post-operative period time-point. HR and non-invasive blood pressure were recorded while performing TTE in the immediate pre-hospital discharge period. Stroke volume was calculated using formula: $\pi/4 \times (\text{LVOT Diameter})^2 \times \text{LVOT VTI. Cardiac}$ output was calculated as: Stroke volume × HR. Stroke Index and C.I were calculated by dividing the stroke volume and cardiac output by BSA, respectively. Average value arising out of three consecutive cardiac cycles was taken as the measured value. Systemic vascular resistance index (SVRI) was calculated using the formula: $SVRI = [80 \times (MAP-CVP)]/C.I.$ CVP was estimated at the immediate pre-hospital discharge time point from the inspiratory and expiratory diameter of CVP, as described by ASE.^[12]

Sample size calculation

As we were unaware about any published data that clearly specify the magnitude of change in peak LV GLS in the perioperative period that is clinically significant or of concern, considering that a 20% difference between the peak LV GLS values at the two time points of our study as significant, the effect size was derived as 0.44. Based on this effect size and considering an alpha error level of 0.05 and 80% power, a sample size of 42 was derived for paired *t*-test. Expecting a dropout of 20%, a sample size of 50 was required for the study.

Statistical analysis

All the values of the echocardiographic, demographic, hemodynamic, ventilatory, anesthetic, and the other clinical

parameters data were checked for normal distribution by Kolmogorov–Smirnov test and also by visual Q–Q plotting. The values of the normally distributed parameters were presented as mean (\pm Standard deviation [SD]) and the values of the parameters not following normal distribution were presented as median (interquartile range).

The parameters having normal distribution were compared between the immediate post-operative and immediate pre-hospital discharge time points using paired t-test. The parameters not distributed normally were compared between the two time points using the Wilcoxon test. The change in peak LV GLS from the immediate post-operative period to the immediate pre-discharge period was compared among patients with normal versus abnormal pre-induction peak LV GLS, respectively, using Welch's t-test. The association between the change in left ventricular peak GLS and C.I from immediate post-operative period to immediate pre-hospital discharge period and the vasoactive inotropic score (VIS) at the immediate post-operative period was estimated visually by scatter plot and subsequently with linear regression technique with calculation of R² values. The association of left ventricular peak GLS and C.I at the immediate post-operative period with the VIS at the immediate post-operative period was also estimated similarly.

The patients were subdivided in 3 subgroups according to tertiles of VIS at the immediate post-operative period. The change in peak LV GLS and C.I from the immediate post-operative period to the immediate pre-hospital discharge period was compared using ANOVA in between the patients of the 3 groups created by the VIS-tertiles.

Reliability of echocardiographic measurements was evaluated using the intraclass coefficient (ICC) from a two-way mixed-effects model with absolute agreement. Interobserver variability was tested by performing a review and analysis of the saved images of a random 15 patients by another experienced echocardiographer. Intra-observer variability was tested by repeating analysis of the saved images of a random 15 patients by the same observer who had taken readings initially, 1 month after the initial analysis. Good reliability was considered to be indicated by an ICC > 0.80.

A P < 0.05 was taken as statistically significant difference. All the statistical analyses were performed using the Statistical Package for the Social Sciences (IBM) 16.0 software.

RESULTS

Among eighty-eight (88) patients approached for the study, the study could be successfully completed as well as analyzed in fifty-one (51) patients [Figure 1]. The demographic parameters, comorbidities, pre-operative and pre-discharge medications, surgical and CPB characteristics, baseline (preinduction) echocardiographic and hemodynamic parameters are summarized in Table 1.

Pre-induction Peak LV GLS (Mean [SD]) and LVEF (Median [IQR]) was 14.5 (4.9)% and 58 (55-64)%, respectively [Table 1]. There were statistically significant differences in Peak LV GLS, C.I, LVEF, HR, and MAP between the immediate post-operative period and prehospital discharge period [Table 2 and Figure 2]. However, There was no statistically significant difference of change in Peak LV GLS from Immediate post-operative period to prehospital discharge period between patients with Normal (n = 17) and Reduced (n = 34) baseline (pre induction) Peak LV GLS (Mean [SD] difference -1.8 (3.9)% vs. -4.1 (4.8)%, P = 0.079, Welch's *t*-test). There was statistically significant, but weak association of the baseline peak LV GLS with the change in Peak LV GLS from immediate postoperative period to the pre-hospital discharge period ($R^2 = 0.156$, P = 0.003 [Figure 3]. However, post-operative VIS was not found to be significantly associated with the change in Peak LV GLS from the immediate post-operative period to pre-hospital discharge period ($R^2 = 0.003$, P = 0.696) [Figure 4].

Based on the VIS score in the immediate post-operative period, the patients were divided into three equal-sized subgroups according to VIS-tertiles – Low (Median 0.09 [IQR 0.00–1.33]), Intermediate (Median 4.62 [IQR 3.43–5.13]), and High (Median 12.8 [7.7–17.5]).



Figure 1: A schematic flow diagram summarizing the selection of the study population LVEF: Left ventricular ejection fraction, MR: Mitral regurgitation, CABG: Coronary artery bypass grafting, POD: Post-operative day, TTE: Transthoracic echocardiography.

 Table 1: Demographic parameters, co-morbidities, pre-operative and pre-hospital discharge medications, coronary artery territories diseased and re-vascularized, baseline (pre-induction) echocardiographic and hemodynamic parameters, intra-operative parameters of the study population.

 Parameters

Demographic				
Age (years)	56.5 (7.7)*			
Gender (M/F)	43/8			
Height (m)	1.63 (0.09)*			
Body weight (kg)	65.9 (10.3)*			
Comorbidities				
Hypertension	26 (51%)*			
DM	05 (10%)*			
Hypothyroidism	01 (1.96%)#			
HTN and DM	14 (14 (27.45%)#		
HTN, DM, and hypothyroidism	01	01 (1.96%)#		
Chronic kidney disease	01 (1.96%)#			
Medications	Pre-operative	Pre-hospital discharge		
Beta blocker	41 (80%)#	44 (86%)#		
ACE inhibitors/ARB	22 (43%)#	12 (24%)#		
Diuretics	17 (33%)#	37 (73%)#		
Antianginal	49 (96%)#	43 (86%)#		
Antihypertensive	18 (35%)#	20 (39%)#		
Antidiabetic	17 (34%)#	18 (35%)#		
Statins	37 (73%)#	51 (100%)*		
Thyroxine	2 (4%)#	2 (4%)#		
Antiplatelets	21 (41%)#	51 (100%)#		
Antiarrhythmic	-	2 (4%)#		
Coronary artery territories involved	Diseased	Re-vascularized		
LAD	51 (100%)*	51 (100%)*		
LCX	44 (86%)#	42 (82%)#		
RCA	40 (78%)#	40 (78%)#		
Pre-induction echocardiographic and hemodynamic parameters				
Peak LV GLS (%)	-14	-14.5 (4.9)*		
LVEF (%)	$58~(55{-}64)^{\wedge}$			
IVC diameter (Inspiration) (cm)	$1.2 (1.1-1.4)^{\wedge}$			
IVC diameter (Expiration) (cm)	1.8 (1.7–1.9)^			
CVP	9 (8–10)^			
HR	75 (62–83)^			
MAP	80 (74–87)^			
Intra-operative parameters				
CPB Duration (min)	138.3 (39.2)*			
AXC Duration (min)	97.4 (27.4)*			
Min. Temp (°C)	32.8 (1.1)*			

*Described as Mean (SD), *Described as Number (Percentage), ^Described as Median (IQR). HTN: Hypertension, DM: Diabetes mellitus, ACE: Angiotensin converting enzyme, ARB: Angiotensin receptor blocker, LAD: Left anterior descending artery, LCX: Left circumflex coronary artery, RCA: Right coronary artery, LV GLS: Left ventricular global longitudinal strain, LVEF: Left ventricular ejection fraction, IVC: Inferior vena cava, CVP: Central venous pressure, HR: Heart rate, MAP: Mean arterial pressure, SD: Standard deviation, IQR: Inter-quartile Range, CPB: Cardiopulmonary bypass, AXC: Aortic cross clamp

Table 2: Difference between parameters at immediate post-operative period and pre-hospital discharge period.								
Parameters	Immediate postoperative period	Pre-hospital discharge period	Mean difference	95% CI	P-value			
Peak LV GLS (%)	-12.8 (3.8)	-10.2 (2.4)	2.6	1.4-3.7	0.000*			
LVOT VTI (cm)	18.5 (4.4)	17.7 (4.3)	0.9	-0.3 - 1.9	0.131			
SI (ml.beat ⁻¹ .m ⁻²)	35.3 (9.4)	33.9 (9.8)	1.4	-0.7 - 3.5	0.183			
Cardiac Index (L.min ⁻¹ .m ⁻²)	3.1 (0.8)	2.5 (0.9)	0.6	0.4-0.8	0.000*			
LVEF (%)	56.4 (53-62)	58.9 (56-64.1)	-	-	0.004*			
LVEDA (cm ²)	18.1 (15.4–19.2)	18.2 (16.1–20.1)	-	-	0.973			
HR (bpm)	88 (84–92)	68 (68-82)	-	-	0.000*			
MAP (mmHg)	68 (64–78)	85 (81–92)	-	-	0.000*			
CVP (mmHg)	8 (8–10)	8 (8–15)	-	-	0.006*			
SVRI (dynes.m ² .cm ⁻⁵)	1574 (1301–2115)	2531 (1985–3542)	-	-	0.000*			

Paired *t*-test for normally distributed variables, Wilcoxon test for non-normally distributed variables, **P*<0.05 considered as statistically significant). LV GLS: Left ventricular global longitudinal strain, LVOT VTI: Left ventricular outflow tract velocity time integral, SI: Stroke Index, CI: Confidence interval, LVEF: Left ventricular ejection fraction, LVEDA: Left ventricular end-diastolic area, IVC: Inferior vena cava, SVRI: Systemic vascular resistance index, HR: Heart rate, MAP: Mean arterial pressure, CVP: Central venous pressure, SVRI: Systemic vascular resistance index



Figure 2: Boxplot showing comparison between immediate post-operative and pre-hospital discharge peak LV GLS (The horizontal bars represent the median values; the upper and the lower margins of the boxes represent the 1st and the 3rd quartiles, respectively; the top and the bottom whiskers represent the minimum and the maximum values, respectively. X represents Mean values). (*P < 0.05 in comparison to Immediate Post-operative Peak LV GLS). LV GLS: Left ventricular global longitudinal strain.

Although VIS score was significantly different between the VIS tertiles (*P*-value 0.000 with Friedman's test), change of Peak LV GLS from Immediate Postoperative to Pre-Hospital Discharge period, Peak LV GLS in the immediate postoperative period, Change in C.I from Immediate Postoperative to Pre Hospital Discharge period, C.I in the immediate postoperative period were not significantly different between the subgroups of patients according to these VIS-tertiles [Table 3].



Figure 3: Scatterplot with superimposed linear regression line showing association of change in Peak LV GLS from immediate post-operative period to pre-hospital discharge period with the baseline (pre-induction) Peak LV GLS (absolute value). LV GLS: Left ventricular global longitudinal strain.

The inter- and intra-observer agreements of the echocardiographic measurements at both the Immediate Postoperative period and the Pre Hospital Discharge period showed that except LVEF, all other parameters had acceptable (ICC>0.8) inter-observer as well as intra-observer agreement at both the timepoints.

DISCUSSION

In our patients, although LVEF improved statistically significantly before hospital discharge in comparison to the immediate post-operative period (56.4 (53–62)% to 58.9 (56–64.1)%, P = 0.011), the peak LV GLS showed a

statistically significant decrease over the same time period $(-12.8 \ [3.8]\%$ to $-10.2 \ [2.4]\%$, P = 0.000). However, LVEF values in both immediate post-operative, as well as pre-discharge period were > 50% and the difference in LVEF between these two time points was only 2.5%. Hence, the difference in LVEF between these time points was clinically insignificant.

Myocardial strain analysis is a more sensitive marker of global as well as regional LV dysfunction than LVEF. Reproducibility and inter-observer agreement of Peak LV GLS are better than those of LVEF calculated by Simpson's method.^[13] Two-thirds of our patients had impaired pre-operative Peak GLS despite having normal pre-operative LVEF. This is consistent with existing literature that a significant number of CAD patients scheduled for CABG have impaired Peak GLS despite having normal LVEF.^[14,15] Lack of improvement



Figure 4: Scatterplot with superimposed linear regression line showing association of change in Peak LV GLS from immediate post-operative period to Pre-Hospital discharge period with VIS at immediate post-operative period. LV GLS: Left ventricular global longitudinal strain, VIS: Vasoactive-inotropic score.

in GLS after coronary revascularization is associated with negative LV remodeling.^[16] While a few studies have reported improvement in LV GLS after CABG,^[17] a few have reported decrement, too.^[14] However, most of the studies have assessed GLS after 1 to 24 months of CABG.

Difference of GLS between pre-operative baseline state and postoperative follow-up is mainly dependent on reverse remodeling of myocardium due to revascularization, which may continue to evolve over months.^[18] Perioperative period, on the other hand, is more dynamic. Changes in Peak LV GLS within the span of the perioperative period of CABG surgery can be affected by a multitude of perioperative factors, like effect of anesthetic agents, altered loading conditions, positive pressure ventilation, ischemia-reperfusion injury, CPB, cardioplegia, vasopressors, and/or inotropes.^[8] Perioperative echocardiographic assessment of cardiac function is a quintessential part of cardiac anesthesiology and intraoperative assessment of LV function is predominantly dependent on TEE.^[19]

The peak LV GLS measured intraoperatively by TEE immediately after CABG, which is used to quantify post-CPB left ventricular function, is expected to change at the time of the patient's discharge, when the perioperative factors influencing LV function are absent. This magnitude of change in peak LV GLS in patients receiving a standardized perioperative management and having an uneventful perioperative course describes the course of peak LV GLS in the immediate postoperative period. Although a few studies have investigated the change of peak LV GLS in the perioperative period, the change in LV function over the early post-operative period has not been addressed adequately. Our study fills this particular knowledge gap in literature. This knowledge for making prediction regarding pre-discharge LV function of a person undergoing CABG and having uncomplicated early post-operative course, as pre-discharge TTE is often not feasible in a busy tertiary care

Table 3: Comparison of change of Peak LV GLS and C.I from the immediate postoperative period to pre-hospital discharge period and the peak IV GLS and C.I in the immediate post-operative period between subgroups in low, intermediate, and high VIS-tertiles, respectively.

Parameters	Low VIS tertile (0.09 [0.00-1.33]) (<i>n</i> =17)	Middle VIS tertile (4.62 [3.43-5.13]) (<i>n</i> =17)	High VIS tertile (12.8 [7.7–17.5]) (<i>n</i> =17)	<i>P</i> -value (One-way ANOVA)			
Change of peak LV GLS from immediate post-operative to pre-hospital discharge period	-2.5 (5.3)	-2.9 (3.7)	-2.3 (3.9)	0.902			
Peak LV GLS in the immediate post-operative period	-12.5 (4.5)	-13.6 (2.9)	-12.2 (3.9)	0.508			
Change in cardiac index from immediate post-operative to pre-hospital discharge period	-0.7 (0.7)	-0.5 (0.7)	-0.5 (0.8)	0.672			
Cardiac index in the immediate post-operative period	3.1 (0.8)	3.0 (0.6)	3.1 (0.9)	0.903			
One way ANOVA BCODE considered statistically significant LV CLS. Left ventricular global longitudinal strain VLS. Vacanstive instrumic score							

One-way ANOVA, *P*<0.05 considered statistically significant. LV GLS: Left ventricular global longitudinal strain, VIS: Vasoactive-inotropic score, ANOVA: Analysis of variance, C.I: Cardiac index

hospital due to the enormous workload on the cardiology department and also due to poor transthoracic imaging window in a significant number of patients in the early post-operative period.

A multitude of reasons can be attributed to the significant deterioration of peak LV GLS from the immediate postoperative period after CABG (measured just before shifting from Operating Room [OR] to Intensive Care Unit [ICU]) to that just prior to the hospital discharge, despite having uneventful intraoperative and post-operative course. The intraoperative measurement of peak LV GLS was done with TEE after completion of CABG and sternal closure, just before shifting to ICU. The patients were under anesthesia with a median BIS of 42, were on positive pressure ventilation, and were on vasoactive-inotropic support with a median VIS of 4.8. Moreover, they were shortly weaned off from CPB after CABG, therefore, still under the immediate effects of myocardial revascularization, ischemia-reperfusion effect of aortic clamping and unclamping, systemic effect of CPB as well as residual effect of cardioplegia. On the other hand, pre-discharge peak LV GLS was assessed using TTE, patients were awake and spontaneously breathing, not under any vasoactive-inotropic support and a median of 9 days had passed since they had undergone surgery, therefore, the immediate effects of CPB, ischemia-reperfusion injury and cardioplegia had likely weaned off by that time although these changes can continue to evolve over time.^[20]

Labus *et al.*^[8] found significant decrease of 2D LV GLS from pre-operative baseline to pre-hospital discharge period after on-pump CABG with the uneventful perioperative course, although LVEF remained unchanged. We also found a similar decrease in 2D LV GLS from pre-induction baseline to the pre-hospital discharge period, with no clinically significant difference in LVEF. Existing studies also suggest that 2D-LV GLS deteriorates after on-pump cardiac surgery as well as after off-pump CABG.^[14,18,21]

Our study adds further to the literature by gaining insight into the change of peak LV GLS from immediate postoperative period after CABG to the pre-hospital discharge period, thus quantifying the change of LV function over the dynamic early post-operative period. As we chose patients who had uneventful surgery with uneventful intraoperative as well as postoperative period, the change in peak LV GLS over this time period arguably represents the usual change of LV function that occurs over this early post-operative period with gradual attenuation of the intraoperative factors that were present during the intraoperative assessment of LV function immediately after CABG.

Labus *et al.*^[8] did not find any significant difference between TTE-derived peak LV GLS obtained in awake, spontaneously breathing state and TEE-derived peak LV GLS obtained in anesthetized state with positive pressure ventilation

in a similar population of patients like ours. Although TTE-based studies like study by Dalla *et al.*^[7] have found significant deterioration of peak LV GLS upon induction of anesthesia and onset of positive pressure ventilation, their study population was devoid of any known myocardial disease. Cinotti *et al.*,^[22] in their study involving non-cardiac population had found improvement in peak LV GLS after cessation of general anesthesia and extubation. Based on these, it can be argued that the deterioration of the peak LV GLS from immediate post-operative period after CABG to the pre-hospital discharge period was not contributed significantly by the transition from anesthetized state with positive pressure ventilation to awake, spontaneously breathing state.

Peak LV GLS is also affected by inotropic and vasopressor therapy through their effect on myocardium and the loading conditions.^[23] Unlike the study of Labus et al.^[8] where peak LV GLS was measured intraoperatively by TEE once before and once after sternotomy before institution of CPB, without any influence of vasopressor or inotropic support, peak LV GLS was measured in our study intraoperatively after CABG was completed and after weaning from CPB, a period where vasoactive or inotropic support is almost unavoidable. LV strain has been found to increase dose dependently with the use of inotropic agents.^[23] We did not find any significant linear relation between the magnitude of the decrement of peak LV GLS from the immediate postoperative period after CABG to the pre-hospital discharge period and the VIS at the immediate post-operative period after CABG. The finding was further supplemented by comparing the decrement of peak LV GLS among the patients receiving low, intermediate and high vasoactive-inotropic support at the immediate postoperative period after CABG, where no significant difference was found among the patients in low, intermediate, and high VIS tertiles. This lack of association can possibly be explained by the fact that vasopressors and inotropes are used in the immediate post-operative period after cardiac surgery to counteract the effects of revascularization, CPB, aortic cross clamp (AXC), and cardioplegia on the myocardium and vasculature to achieve and maintain adequate cardiac output as well as systemic perfusion pressure.^[24] The effects of the intraoperative factors on the myocardium and vasculature vary from patient to patient leading to different amounts of dysfunction of myocardium and vasculature on different patients. The more the dysfunction, the more is the requirement of the vasoactive-inotropic drugs to maintain adequate cardiac output and/or systemic perfusion pressure. Therefore, the measurements made at the immediate postoperative period after CABG represent a balanced state of counteraction between at one side the factors that depress LV function and the factors that enhance LV function at the other side. Due to the same reason, the magnitude of change of peak LV GLS as well as C.I did not have an association

with the VIS score at the immediate post-operative period. Although based on the findings of our study, we cannot definitely say that the deterioration of peak LV GLS was not contributed by the presence and absence, respectively, of vasoactive-inotropic support, we can say that the magnitude of the decrement in peak LV GLS was not associated with the level of vasoactive-inotropic support present at the immediate post-operative period after CABG.

Impaired peak LV GLS in the pre-operative period is an independent predictor of poor prognosis after cardiac surgery,^[15] over and above LVEF.^[25] In our study, the deterioration of peak LV GLS from the immediate post-operative period to pre-hospital discharge period was not significantly different between patients having normal and impaired pre-operative peak LV GLS and the decrement in peak GLS was also found to be weak. However, as the sample size of our study was not calculated to look at this association specifically, a definitive conclusion regarding the same cannot be drawn from our findings.

Peak LV GLS is dependent on loading conditions on heart at the time of measurement.^[26] Peak LV GLS increases with increase in preload and decreases with decrease in preload.^[27] There was no significant difference in LVEDA between the two time points. CVP estimated by IVC diameter and variability at the time of hospital discharge was higher than the directly measured CVP at the immediate post-operative period after CABG. Therefore, the preloads at the two time-points were arguably similar. Although comparison of pulmonary capillary wedge pressure measured through a pulmonary arterial catheter between the two time points would have been the gold standard, it is not feasible for a patient to have pulmonary arterial catheter at the time of hospital discharge. Therefore, based on our findings, it is to a reasonable extent unlikely that the decrement of peak LV GLS was related to preload.

The afterload, estimated by SVRI, was significantly higher at the time of hospital discharge than at the immediate postoperative period after CABG. This could potentially be a significant contributor toward the decrement of peak LV GLS, as increase in afterload is associated with decrease in peak LV GLS. Lower afterload immediately after CABG in comparison to that at the time of hospital discharge can possibly be explained by presence of general anesthesiainduced dilation of systemic resistance vessels,^[28] reduced transmural pressure due to positive pressure ventilation,^[29] use of inodilator agent (milrinone), and vasoplegia after CPB at the intraoperative period immediately after CABG.^[30] Although no significant linear relation was found between the change in peak LV GLS as well as C.I and the change in SVRI, the lack of linear relation can be explained by the complex interplay and inter-dependency of the loading

conditions and myocardial contractility in the loop-type human circulatory system.

There were certain strengths of our study, the intraoperative as well as the post-operative management of our patients was essentially uniform, according to a pre-standardized protocol. All the surgeries were performed by a single surgeon, using essentially the same surgical technique. The techniques of myocardial preservation were also similar among the patients. The CPB and AXC durations, with a mean of 138 min and 97 min, respectively, were not prolonged. The pre-operative risk status of the patients, with a median (IQR) Euroscore II of 0.87 (0.78-0.99), was low. Only the patients with an uneventful, uncomplicated intraoperative as well as postoperative course till hospital discharge were included. Hence, findings of our study essentially represent the usual course of change of LV function from that at the immediate postoperative period, to that at the time of hospital discharge, in low-risk patients with normal pre-operative LV function (by LVEF) undergoing uneventful, multivessel CABG with uneventful early post-operative course. Thus, our findings shed light into the change of LV function over the early post-operative course till the time of discharge from the hospital after CABG a scarcely investigated domain till now. The findings of our study can form the base upon which course of change of LV function in this early post-operative period in highrisk patients, patients with pre-operative impaired LVEF, and patients having not-so-benign intraoperative and/or early post-operative course after CABG can be studied.

The association between the change of peak LV GLS over this early post-operative period with the likely perioperative factors that can affect peak LV GLS values was also looked upon in our study, although probably underpowered for the same. With the help of our findings, adequately powered studies can be designed in the future to understand the contribution of different perioperative factors in affecting the change of LV function over this scarcely investigated early post-operative period after CABG.

Limitations

The two time points chosen for quantifying the change of peak LV GLS over the early post-operative period were a median of 9 days apart and represented LV function at those two particular points only. Serial measurements of peak LV GLS, which would have detected the course of the LV function in the dynamic early postoperative period were not done. However, the feasibility of obtaining good quality transthoracic echocardiographic images for the purpose of accurate measurement of peak LV GLS is poor in the early post-operative period due to the presence of chest as well as pericardial drains and retained intrapericardial and intrapleural air.^[31] Therefore, the period before hospital

discharge was chosen, when all the drains had been removed and only patients in whom optimum image could be obtained for accurate measurement of peak LV GLS were included.

Immediate post-operative GLS and pre-hospital discharge GLS were measured using different modalities, i.e., by TEE and TTE, respectively. However, this reflects usual practice as TEE is the mainstay modality in the intraoperative period of cardiac surgery and TTE is the mainstay as well as more feasible modality in situations other than intraoperative period. Moreover, TEE-measured GLS has been shown to be an appropriate alternative to TTE due to good agreement.^[32-34]

Findings of our study may not be extrapolated to patients having high perioperative risk status, reduced pre-operative LVEF, and/or who had complicated intra- as well as postoperative course. As already discussed, our study was not powerful enough to find the presence or absence of an association of various perioperative factors with the change of peak LV GLS from that in the immediate postoperative period to that at the time of hospital discharge definitively. Therefore, no analysis could also be undertaken to understand whether any particular subgroup was affected more by the perioperative factors. Further studies can be designed for this purpose with the help of our findings.

Due to time-bound nature of the study, no subsequent measurement of peak LV GLS could be done and whether the decline of peak LV GLS over this early post-operative period after CABG has any prognostic significance could not be assessed. Further studies are required to specifically address this aspect.

CONCLUSION

Low-risk patients with normal LVEF undergoing multivessel CABG surgery with uneventful intraoperative and early postoperative (during hospital stay) course have significant decline in peak LV GLS from the immediate intraoperative period (immediately before shifting from OR to ICU) to the time just before their discharge from hospital, despite having no clinically significant difference in LVEF between the two time points. Increase in afterload explains in part this decline of peak LV GLS over this time period.

Further studies with adequate power are required to find whether the decrement of peak LV GLS over the early postoperative period bears any prognostic significance, which can, in turn, direct the need for routine measurement of peak LV GLS in the perioperative and early post-operative period in patients undergoing CABG.

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