Proceeding S.Z.P.G.M.I. Vol: 27(2): pp. 61-68, 2013.

Effects of Coronary Artery Bypass Grafting on QT Dispersion

Muhammad Saleem Khan Abbasi, Qazi Abdul Saboor, Saulat Siddique and Amber Malik
Department of Cardiology, Shaikh Zayed Postgraduate Medical Institute, Lahore

ABSTRACT

Abnormal dispersion of QT interval (QTd) measured as the interlead variability of QT, reflects an in homogeneity of ventricular action potential. This is increased in coronary artery disease (CAD) due to ischemia of myocardium. **Aims and Objectives:** To determine / investigate the values of QT dispersion in our population. To investigate whether there is any short term influence of coronary artery bypass grafting (CABG) on QT dispersion, evaluated early (7 days) after surgery. **Design of study:** Single center observational study. **Site of study:** Department of Cardiology & Cardiothoracic Surgery, Shaikh Zayed Hospital, Lahore. **Patients and Methods:** A total of 50 consecutive patients, who underwent CABG in Shaikh Zayed Hospital, were included in the study. All the patients were given the standard therapeutic treatment for the management of ischemic heart disease (IHD). Biochemical lab data including complete blood count, blood sugar level, serum electrolytes, urea & creatinine was collected and assessed for any abnormality before the operation. A twelve lead electrocardiogram (ECG) was recorded during a resting period in all patients planned for CABG 24 hours before surgery. ECG was repeated 48 hours after operation and at the time of discharge from the hospital. QT interval was calculated from the onset of QRS complex to the point of return of 'T' wave to isoelectric line or to the nadir between 'T' wave and 'U' wave, in cases where a 'U' wave was present. QT dispersion was measured as the difference between maximum and minimum QT from the 12 lead ECG. **Results:** The mean QTd in healthy individuals was (53.60±19.56 ms) (p=0.000). There was significant reduction in QTd from 24 hours before CABG to 48 hours and seven days after CABG (74.80±28.59 ms) (p=0.000) vs. (46.40±17.82 ms) (p=0.000) and (33.20±15.44 ms) (p=0.000) respectively. **Conclusion:** There is a significant reduction of QTd early after CABG. It seems that QTd is a simple non-invasive and a reliable mode for detecting coronary ischemia and also for evaluating the effects of revascularization.

Key Words: Coronary Artery Disease (CAD), ischemic Heart Disease (IHD), Coronary Artery Bypass Grafting (CABG), QT dispersion (QTd).

INTRODUCTION

Coronary artery disease (CAD) remains a major health problem in the United States of America and other affluent nations of the world despite advancement in diagnostic skills and treatment modalities over the past several years.[1] Each year almost 700,000 Americans die from complications of CAD and more than one million acute myocardial infarctions (AMI) occur. The economic impact of CAD on American society exceeded $90 billion in 1997. Although the death rate from acute myocardial infarction declined by about 30% over the past decade, its development is still a fatal event in approximately one third of the patients. According to the World Heart Federation, the incidence of CAD in the developing countries is increasing rapidly and soon will be more closely aligned with those now afflicting developed countries.[2-3] The prevalence of ischemic heart disease (IHD) due to atherosclerosis is increasing in Pakistan as in other developing countries and

coronary artery mortality is also increasing in Pakistan. Atherosclerosis, a major cause of CAD, is a progressive disease that most often produces clinical symptoms in middle to late adulthood. Depending on the severity of underlying lesions and biological triggers, a spectrum of clinical syndromes ranging from stable angina pectoris to acute coronary syndrome and ischemic sudden death may occur. A steady fall in the mortality rate from myocardial infarction has been observed across several population groups. This appears to be caused by a fall in the incidence of acute myocardial infarction (replaced in part by an increase in rate of unstable angina) and a fall in the case fatality rate once an AMI has occurred.

Several phases in the management of patients with CAD have contributed to the decline in the mortality from CAD. The "clinical observation phase" of coronary care consumed the first half of the 20th century and focused on a detailed recording of physical and lab findings. Treatment consisted of strict bed rest and sedation. Subsequently the "coronary care unit phase" beginning in the mid 1960s occurred and was notable for detailed analysis and vigorous management of cardiac arrhythmias.

The "high technology phase" was started by the introduction pulmonary artery balloon flotation catheter, setting the stage for beside hemodynamic monitoring and more precise management of heart failure and carcinogenic shock associated with CAD.

The modern "reperfusion era" of coronary care was introduced by intracoronary and then intravenous thrombolytic, increased use of aspirin and development of coronary revascularization modalities. CABG preceded percutaneous coronary angioplasty and has had a remarkable success in alleviating symptoms and improving lifestyle and has improved mortality rate in selected patients.

There are various non-invasive tests for the diagnosis of the CAD. Recently it has been observed that, "QT dispersion", an interlead difference of QT interval measured on a 12 lead ECG, is a marker of coronary ischemia and there is convincing evidence that coronary ischemia augments QT dispersion.

AIMS AND OBJECTIVES

Purpose of study

The specific aims & objectives of study are as follows:

- To investigate the values of QT dispersion in our population.
- To investigate whether there is any short term influence of CABG on QT dispersion, evaluated early (7days) after surgery.

PATIENTS AND METHODS

Study design

Single centre prospective observational study

Site of study

Department of Cardiology & Cardiothoracic Surgery, Shaikh Zayed Hospital, Lahore.

Selection of patients

A total of 50 patients were selected. Following criteria was adopted to select the patients.

Inclusion criteria

All patients undergoing CABG in Shaikh Zayed Hospital, Lahore, were included in the study.

Exclusion criteria

All patients with intraventricular conduction defects, pre-excitation, treatment with digoxin or other antiarrhythmic drugs, electrolyte imbalance and history of congenital long QT syndrome were excluded from the study.

Selection criteria for healthy individuals

A total of fifty age and sex matched healthy individuals were included in the study.

Inclusion criteria

Fifty healthy individuals having no evidence of any major systemic illness and having normal ECG were included in the study.

Exclusion criteria

All patients with intraventricular conduction defects, pre-excitation, treatment with digoxin or other antiarrhythmic drugs, electrolyte imbalance

and history of congenital long QT syndrome, IHD and diabetes mellitus were excluded from the study.

Statistical analysis

All data was transferred from performa to computer using SPSS 22. The epidemiological features and risk factors were evaluated. Numerical data was reported as frequency & percentage. Numerical variables were recorded as Mean±S.D. QT dispersion of patients 24 hours before CABG was compared with 48 hours and 7 days after CABG by using ANOVA. QT dispersion of healthy individuals was compared with QT of patients 24 hours before CABG by using unpaired two tailed T test. A p-value of ≤ 0.05 was considered significant for all analysis.

RESULTS

Among 50 patients included in the study, 33 were male and 17 were female (Table 1). Ages ranged between 33-70 years. The total number of patients having hypertension was 28%, diabetes mellitus 58%, smokers 68%, previous history of IHD 46% and family history of ischaemic heart disease (FH^+) 74%.

All the patients undergoing CABG remained asymptomatic during recovery periodand first week post CABG with no evidence of residual ischaemia on history and lab investigations. A total of fifty age and sex matched healthy individuals were also included in the study for the evaluation of QT dispersion in our healthy population.

The mean QT dispersion in our healthy control group was 53.60±19.56 ms. There was a trend towards higher values of QT dispersion in younger age group from 35-55 years, (58.50±4.20 ms) as compared to those having age >55 years (48.70±4.58 ms) except for two patients with age above 70, who had QT dispersion of 80 ms.

Among healthy females (17), mean value of QTd was 49.41±21.35 ms (p=0.000) as compared to their male counter parts with mean value of QTd of 55.76±12.33 ms (p=0.000).

Among the patients, QTd calculated from 12 lead ECG done 24 hours before CABG was 74.80±28.59 ms (p=0.000). 48 hours after CABG, mean QTd dropped to 46.40±17.82 ms (p=0.000), which further reduced to 33.20±15.44 ms (p=0.000) when estimated from ECG done 7 days after CABG (Table 2).

Table 1: Gender distribution

Gender	No. of patients	Percentage (%)
Male	33	66
Female	17	34
Total	50	100

Table 2: QTd before and CABG.

Time of estimation	QTd values	P value
24 Hour before CABG	74.80±28.59 ms	0.000
48 Hour after CABG	46.40±17.82 ms	0.000
7 days after CABG	33.20±15.44 ms	0.000

DISCUSSION

Dispersion of QT interval (QTd) has been proposed as a non-invasive marker for assessment of the inhomogeneity of ventricular repolarization and as a predictor of cardiac electrical instability. It is hypothesized that QTd results from myocardial fibrosis and ischemia, neurohormonal activation, electrolyte and metabolic imbalance and the influence of drugs. Recently, it has been shown that QT dispersion increases reversibly during ischemia in patients with coronary artery disease.[4-5] It could be expected that surgical revascularization as a treatment of patients coronary artery disease may be an intervention which leads to a decrease in the dispersion of repolarization.

Roukema et al [6] observed that QTd at peak exercise was greater in the CHD group than in a control group. They also found that beta blockers had a blunting effect on exercise related changes in QTd.

Over the past several years, numerous clinical studies have been conducted evaluating the effects of coronary ischemia on disparity of ventricular repolarization as assessed by determination of QT dispersion from the surface ECG. From findings in patients with acute MI, stable CAD and vasospastic angina, there is convincing evidence that acute coronary ischemia augments QT dispersion.[7-8]

In some studies, this was associated with stress induced myocardial ischemia on peak exercise and early recovery during treadmill exercise electrocardiogram of patients even without exercise induced chest pain or ST depression and was suggested as a useful indicator of coronary artery stenosis independent of gender and/or exercise induced ST segment depression.[9]

Yoshimura M et al. conducted a study in Japan on 50 patients, who underwent exercise stress test for the evaluation CAD. Exercise ECGs were analyzed for QTd. All the patients underwent coronary angiography for clinical indications. None of them showed ST segment depression during or after exercise. There were 25 patients with significant CAD and 25 without significant CAD on coronary angiography.

The QTd measured before, immediately after and one minute after exercise was similar in the two groups. The QTd at three and five minutes after exercise was significantly greater in patients with CAD than in those without significant CAD and the most marked difference in Qtd was observed at three minutes after exercise. They concluded that QTd at three minutes after exercise of >60 ms has a sensitivity of 80% and specificity of 88% regarding the diagnosis of CAD.

Koide Y et al conducted a study in Japan on 273 consecutive patients (190 men & 83 women) without a history of MI who underwent treadmill exercise electrocardiography and coronary angiography for evaluation of angina. Of these, 146 patients had no significant coronary stenosis, 61 had single vessel disease, 56 had multi vessel disease and 10 had had left main stem CAD.

QT dispersion immediately after exercise was significantly greater in patients with significant coronary stenosis than in those without (64±14 vs. 39±14, $p<0.01$). QTd immediately after exercise was significantly more sensitive in men (sensitivity 75%, specificity 85%) and significantly more specific in women (sensitivity 77%, specificity 85%) than exercise induced ST segment depression (men sensitivity 62%, specificity 74%, women sensitivity 81%, and specificity 68%) as an indicator of significant coronary stenosis.

In other studies, the effects of coronary angioplasty on precordial QT dispersion in patients with symptomatic CAD was evaluated in standard 12 lead ECGs performed 24 hours before, 24 hours after and >2 months after PTCA and it was found that QT dispersion decreases significantly after successful coronary artery revascularization and increases with re-stenosis.[10-13]

Younus A et al conducted a study in Canada on 376 consecutive patients with ischemia due to single vessel CAD, without prior MI, who underwent PTCA. They found a mean QTd of 60±90 ms before PTCA, 23±14 ms immediately after PTCA ($p<0.001$ vs. before PTCA).

Symptomatic recurrent ischaemia in 8 patients with documented restenosis increased QTd to 56±15 ms ($p<0.001$) vs. 25±14 ms immediately after PTCA which decreased again after successful repeat PTCA (22±13 ms, $p<0.10$ vs. before the second PTCA). They concluded that QTd decreases after successful coronary artery revascularization and increase with restenosis.

Additionally, prognostic value of QT interval prolongation in patients with unstable angina was studied which showed that QT interval prolongation is an independent risk predictor in patients with unstable angina of the composite endpoint (death, MI and need for urgent coronary angioplasty or CABG) during a thirty days follow-up period.[14]

Although the cardioprotective effect of some therapeutic interventions such as coronary angioplasty and beta blockers may be explained by their blunting effects on ischaemia related changes in QT dispersion evaluated early (24 hours) after PTCA but insufficient data on the relation between surgical revascularization and dispersion repolarization is available.[15-16]

In one study, influence of CABG on QT dispersion at rest and during exercise was studied before, six months after and two years after CABG. There was a significant reduction in rest and peak exercise QT dispersion from before CABG to six months and two years after CABG.[17-18] However, despite extensive research, no study was found showing the short term effects of CABG on QT dispersion.

Iwona Wozniak-Skowersk et al conducted a study in Poland on 64 male patients with stable angina who were referred to their hospital for further evaluation and subsequent CABG. They

divided the patients into several groups; with and without previous infarction and with ejection fraction on echocardiography of >40% and <40%.

A symptom limited exercise test according to the standard Bruce protocol was performed before CABG and 6 months and 2 years after CABG. QTD analysis was done using 12 leads of an averaged ECG, which was performed at paper speed of 25mm/sec.

The adjustment of QTd for heart rate (QTdc) was calculated according to Bazett formula. The main result were QTd measured at rest (60±20 ms) and at peak exercise (66±22 ms) before CABG, 6 months after CABG (43±14 ms and 38±11 ms) and 2 years after CABG (45±13 ms and 36±11 ms). At baseline, patients showed significant increase in QTd and QTdc, at rest and peak exercise. Six months and 2 years after CABG, there was a significant decrease in the pre-exercise resting value of all these parameters. Moreover, QTd at peak exercise decreased significantly.

It was further observed regardless of ejection fraction values, QTd and QTdc showed a similar decreasing tendency. They concluded that CABG results in long lasting QTd reduction which is independent of baseline LV contractility and its improvement after CABG.

In this study, QT dispersion in healthy individuals was evaluated to establish its normal values in our population, as there is no local data available on the subject. Moreover, the effect of CABG on QT dispersion was evaluated in a standard 12 lead ECG performed 24 hours before CABG and was compared ECG performed 48 hours after operation and at the time of discharge from hospital (average 7.4), to determine the short term influence of CABG on QT dispersion.

Mean values of QTd (53.60±19.56 ms) in healthy individuals were comparable to values found in previous studies. Literature review shows QT dispersion to vary mostly between 30 & 60 ms in normal subjects,[19-20] although average value of around 70 ms was also reported. In 51 studies, in which QT dispersion was measured in 56 groups with total of 8455 healthy control subjects of various ages (including three studies of children), mean QTd values were found to range from 10.5±10 ms to 71±7 ms.[21-22] The weighted mean±SD from all these studies was 33.4±20.3, while the median was 37 ms. Moreover, most researchers reported a wide overlap of values between normal individuals and different patients groups. Therefore, all values proposed for upper normal limit in healthy subjects have subsequently turned out to be unreliable. For example, the WOSCOPS[23] study included 6595 middle aged men with moderately raised cholesterol but no previous MI.

In a multivariate analysis, an increment of 10 ms in QTd increased the risk foe death of coronary heart disease or non fatal MI by 36% (p=0.0041). Qtd of >44 ms carried an increase risk of 36% (p-0.0034) compared to QTd of <44 ms. On the other hand, cut off level of 44 ms had a sensitivity of only 8.8% with a specificity of 93%.

In the present study, it was observed that QTd was slightly greater in males (mean value 56.76±12.33 ms, p=0.000) vs. females (mean value 49.41±21.35 ms, p=0.000) of same age group. This finding was also similar to previous studies in which they found either marginally greater values in men or no statistically significant difference between the genders.[24-26]

The study also revealed that the values of QTd were higher in younger age group (58.50±4.20 ms) as compared to the older people (48.70±4.58 ms). This observation was also reported in different studies where an age related difference of <10 ms were reported and found to be statistically significant in some studies but not in other.[27-28] For example, in the study of Saveliva[29] et al on more than 1000 healthy subjects, QTd was 29.1±17.8 ms in age group of 17-29 years and 21.7±13.3 ms in the age group of 50-80 years. On the other hand, Macfarlane et al found no significant age differences (QTd of 23.6±7.7 ms, 24.8±8.2 ms, 24.8±8.5 ms and 24.5±9.8ms) in the age groups of <30, 30-4-, 40-50 and >50 years respectively.

In our study, QTd was significantly raised in patients (74.80±28.59 ms, p=0.000) as compared to healthy population (53.60±19.56 ms), which was also reported by different researches in the previously described studies, Moreover, QTd in patients 48 hours and 7 days after CABG was markedly reduced as compared to QTd 24 hours Before CABG.[30]

This finding was also similar to a previous

study showing the influence of CABG on QTd, six months and two years after CABG. However, the question why QTd in patients, 48 hours and 7 days after CABG (46.40±17.82 ms & 33.20±15.44 ms, (p=0.000) respectively was significantly lower than healthy controls (53.60±19.56 ms) remains to be answered. In this study also, the QTd was measured by a single observer as in previous studies.

QTd interval is influenced by heart rate and Bazett's formula is used as a correction factor in most studies. It seems reasonable to use this formula for resting heart rates between 60 and 100 bpm. The relationship is, however, influenced by several factors, such as slow adaptation to changes in heart rate, exercise and cardiac drugs.

In the present study, resting dispersion of repolarization was described by QTd. Malik[31] et al have indicated that QT dispersion should be preferred to corrected QT dispersion. Moreover, Zabel[32] et al showed in an experimental study recently that the dispersion of ventricular repolarization did not change significantly with pacing induced changes in cycle length.

CONCLUSION

- The mean QTd in our healthy control group was 53.60±19.56 ms. There was a trend towards higher values of QTd in younger age group (35-45 years) as compared to those having age >55 years.
- On the basis of our analysis, we concluded that dispersion of repolarization is significantly increased in patients with coronary artery disease planned for CABG as compared to healthy individuals. It was further observed that QTd was significantly reduced in patients 48 hours and 7 days after CABG.

REFERENCES

1. Sporton SC, Taggart P, Sutton PM, Walker JM, Hardman Sm. Acute Ischemia: a dynamic influence on QT dispersion. Lancet. 1997;349:306-309.
2. American college of emergency physicians: Clinical policy for the initial approach to adults presenting with chief complaint of chest pain with no history of trauma. Ann Emerg Med. 1995;274-299.
3. Ryan TJ, Antman EM, Brooks NH, et al. ACC/AHA guidelines for the management of patient with AMI. Executive summary and recommendation. A report of ACC/AHA task force on practice guidelines. J Am Coll Cardiology. 1999;34:890-911.
4. Kuo CS, Munakata K, Reddy P, Suaicz B. Characteristics and possible mechanism of ventricular arrhythmia dependent on the dispersion of action potential duration. Circulation. 1983;67:1356:67.
5. FU GS, Meissner A, Simon R. Repolarization dispersion and sudden cardiac death in patients with impaired left ventricular function. Eur Heart J. 1997;18:281-89.
6. Roukema G, Sing JP, Mejis M et al. effect of exercise induced ischemia on QT interval dispersion. AM Heart J. 1998;135:88-92.
7. Lax KG, Okin PM, Kligfield P. Electrocardiographic repolarization measurements at rest and during exercise in normal exercise in subjects and in patients with CAD.
8. Higham PD, Furniss SS, Cambell RWF. QT dispersion and components of the QT interval in ischemia and infraction. Br Heart J. 1995;73:32-36.
9. Koide Y, Yotsukura M, Yoshino H, Ishikawa K. value of QT dispersion in the interpretation of treadmill exercise electrocardiograms of patients without exercise induced chest pain or ST-segment depression. Am J Cardiol. 2000;85:1094-99.
10. Koide Y, Yotsukura M, Yoshino H, Ishikawa K. Usefullness of QT dispersion immediately after exercise as an indicator of coronary stenosis independent of gender or exercise induced ST segment depression. Am J Cardiol. 2000;86:14312-3117.
11. Yunus A, Gills AM, Traboulsi M, Duff JH, Wyse DG, Knudtson ML, Mitchell LB,. Effect of coronary angioplasty on precordial QT dispersion. Med Sci Monit.

2004;10(3):128-31.

12. Yesilbursa D, Serdar A, Aydinlar A. Uludag University, Medical Faculty, Department of Cardiology Bursa Turkey. JpnCirc J. 1999;63(11):881-4.
13. Sedziwy E, Olszowska M, Tracz W, Pieniazek P, Przewlocki T, Woltasik Z. QT dispersion in patient treated with repeated percutaneous transluminal coronary revascularization. Pacing Clin Elcrophysol. 1998;21(11):2407-10.
14. Zhang Y, QiSS, Shen ZQ, Zhou SH. Catheterization Laboratory, Second Xiangya Hospital, Central South University Changsha 410011, China. Effect of percutaneous transluminal coronary angioplasty and stenting on QT dispersion in patients with coronary heart disease. Human Yike Da Xue Bao 2001;26(2):171-2.
15. Shawl FA, Velasco CE, Goldbaum TS, Forman MB.Effects of coronary angioplasty on electrocardiographic changes in patients with unstable angina secondary to left anterior descending artery disease. J AM Coll Crdiol. 1990;16:325-331.
16. Francisco L, Gadaleta MD, Susana C, Liois MD, Alberto R, Lapuente MD. Velislav normal other chambers and valves, Am J Cardiol. 2003;92.
17. Kelly RF, Parillo JF, Hollenburg SM. Effects of coronary angioplasty on QT dispersion. AM Heart. 1997;134:399-405.
18. Trusz-Gluza M, Wozniak-Skowerska I, Rybicka A, Myszor J. Impact of CABG on rest and exercise induced QT dispersion. Eur Heart J. 1997;18:675.
19. Kautzner J, Malik M, QT dispersion and its clinical utility. Pace 1997;20:2625-40.
20. Statters DJ, Malik M, Ward DE, Camm AJ. Qt dispersion: problems of methodology and clinical significance. J Cardiovasc Electrophysiol. 1994;5:672-85.
21. Shah CP, Thakur RK, Reisdorff EJ, Lane E, Aufderheide TP. QTd may be a useful adjunct for detection of myocardial infarction, the chest centre. Am Heart J. 1998;136:496-8
22. Davey PP, Bateman J, Mulligan IP, Forfar C, Barlow C, Hart G. QT interval dispersion in chronic heart failure and left ventricular hypertrophy: relation to autonomic nerves system and holter tape abnormalities. Br Heart J. 1994;71:268-73.
23. Macfarlane PW on behalf of the WOSCOPS. QT dispersion-lack of discriminating power. Circulation. 1998;98(1):81.
24. Tutar H, Ocal B, Imamoglu A, Atalay S. Dispersion of QT and QTc interval in healthy children and effects of sinus arrhythimia on QT dispersion. Heart 1998;80:77-9.
25. Challapalli S, Lingamneni R, Horvath G, Parker M, Goldberger JJ, Kadish A. Twelve lead QT dispersion is smaller in women than in men. Ann Noninvas Electrocardial. 1998;3:25-31.
26. Savelieva I, Camm AJ, Malik M. Gender differences on QT dispersion measured in 1100 healthy subjects. PACE. 1999;22:885
27. Berlu Cl, Sweeten TL, Hill Sl, Vetter VL. Provocative tests in children with suspected congenital long QT syndrome. Ann Noninvas Electrocardiol. 1998;3:3-11.
28. Tutar H, Ocal B, Imamoglu A, Atalay S. Dispersion of QT and QTc interval in healthy children and effects of sinus arrhythimia on QT dispersion. Heart 1998;80:77-9.
29. Savelieva I, Camm AJ, Malik M. Gender differences on QT dispersion measured in 1100 healthy subjects. PACE. 1999;22:885
30. Trusz-Gluza M, Wozniak-Skowerska I, Rybicka A, Myszor J. Influece of CABG on QT dispersion. Med Sci Monit. 2994;10(3):128-131.
31. Malik M, Camm AJ. Mystery of QTc interval dispersion. AML Cardiol. 1997;79:785-87.
32. Zabel M, Woosley Rl. Is dispersion of ventricular repolarization rate dependent? Pacing Clin electrophysiol, 1997;20:2405-11.

The Authors:

Muhammad Saleem Khan Abbasi,
Trainee Registrar
Department of Cardiology,
Shaikh Zayed Postgraduate Medical Institute,
Lahore

Qazi Abdul Saboor,
Assistant Professor
Department of Cardiology,
Shaikh Zayed Postgraduate Medical Institute,
Lahore

Saulat Siddique
Professor
Department of Cardiology,
Shaikh Zayed Postgraduate Medical Institute,
Lahore

Amber Malik
Associate Professor
Department of Cardiology,
Shaikh Zayed Postgraduate Medical Institute,
Lahore

Corresponding Author:

Muhammad Saleem Khan Abbasi,
Trainee Registrar
Department of Cardiology,
Shaikh Zayed Postgraduate Medical Institute,
Lahore