

REGRESSION OF ECG CHANGES AFTER SUCCESSFUL PERCUTANEOUS TRANSMITRAL COMMISSUROTOMY IN PATIENTS WITH SEVERE ISOLATED MITRAL STENOSIS

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Contribution

SZ, TA & ASA conceived the idea, planned the study and drafted the manuscript, collected data, SNHR, TS & NUK did statistical analysis, drafted the manuscript and critically reviewed manuscript. All authors contributed significantly to the submitted manuscript.

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ABSTRACT

Objective: To know regression of ECG changes after successful percutaneous mitral commissurotomy (PTMC) for severe isolated mitral stenosis patients.

Methodology: This cross sectional study was conducted from 1st February 2016 to 31 August 2016 (06 months) in National Institute of Cardiovascular Diseases, Karachi - Pakistan. All consecutive patients admitted for PTMC for severe isolated mitral stenosis were subjected to detailed transthoracic echocardiogram (TTE) and 12 leads ECG before and after procedure as per protocol. All the data were analyzed by SPSS Version 19.0

Results: Among total of 99 study population, females were 65 (65.7%). Mean age was 27.44 ± 6.26 years. Standard 12 lead ECG showed P wave amplitude of 0.26 ± 0.0491 mV pre PTMC versus 0.24 ± 0.036 mV post PTMC ($p < 0.001$). P wave duration in V1/V2 (greater of these two chest leads) was $114.32 \text{ msec} \pm 11.94$ msec (80-140 msec) and $108.64 \text{ msec} \pm 14.55$ (80-140 msec) pre and post PTMC respectively ($p < 0.001$). R wave amplitude in V1/V2 (greater of the two) $0.2879 \pm .2031$ mV vs. $0.2313 \pm .1475$ mV pre and post PTMC respectively ($p < 0.001$). Axis of the heart was 88.28 ± 19.51 degree (45-120) pre PTMC vs. 84.34 ± 19.79 degrees (30-120) post PTMC respectively. RV hypertrophy as demonstrated by R wave in V1 plus S wave in V5 was $0.4830 \pm .2239$ millivolt vs. $0.3980 \pm .1823$ millivolt ($p < 0.001$) pre PTMC & post PTMC respectively.

Conclusion: Successful PTMC procedure causes significant regression of quantitative ECG changes in isolated mitral stenosis patients in mid-term follow up.

Key Words: ECG changes, PTMC, Right ventricular hypertrophy

INTRODUCTION

Rheumatic mitral stenosis is one of the commonest valvular heart lesions in developing countries.¹ Severe mitral stenosis causes various ECG changes which may be considered pathognomonic for this condition. They are P wave abnormality with biphasic morphology, broadening of the P waves and as well as increase in amplitude because of pulmonary hypertension. These changes are more obvious in chest Leads V1, V2 and limb leads II, III & aVF. It may also cause complete or incomplete Right bundle branch block (RBBB) and right axis deviation. Mitral stenosis can lead to enlargement of the left atrium leading to increased risk of thrombus formation.²

In the last two decades; percutaneous transluminal mitral commissurotomy (PTMC) has become the treatment of choice for patients with symptomatic rheumatic mitral stenosis (MS). Several studies have reported good immediate, short-term and long-term results.^{3,4}

It is hypothesized that there may be regression of ECG changes after successful PTMC i.e. right ventricular strain pattern, Biphasic P wave, RBBB/ Incomplete RBBB, R wave in lead V1, axis deviation after mid to long term follow up after successful PTMC.

Since rheumatic heart affection is more severe and the degree of valvular damage is greater in developing countries than in industrialized Western communities, it seems appropriate to examine the frequency, extent and regression of specially the left atrial and right ventricular strain variables on 12 lead ECG in our patients.

The aim of this study was to assess whether changes occur in cardiac parameters as shown by the echocardiographic assessment also have some impact on the electrical activity of the heart by recording and carefully studying their standard ECG in follow up by documenting regression of ECG changes after successful percutaneous mitral commissurotomy (PTMC) in patients with severe isolated mitral stenosis after mid to long term follow-up.

METHODOLOGY

This cross sectional study was conducted from 1st February 2016 to 30th August 2016 in Interventional Cardiology Department of National Institute of Cardiovascular Diseases, Karachi after approval from institution ethical committee.

Severe mitral stenosis was defined by echocardiographic criteria as associated with mean transvalvular gradient of more than 10 mm of Hg, pulmonary artery pressures of more than 50 mm of Hg and a valve area of less than 1 cm².

Successful PTMC was defined as post PTMC mitral valve area of ≥ 1.5 cm² or at least 25 % increase in valve area with no more than one grade increase in MR and with no major

complication (like severe MR, perforation of any cardiac chamber, cerebrovascular accident, major bleeding). Major bleeding was defined any bleeding causes more than 2g of decrease in Hb, intracranial bleeding, intra or retroperitoneal hematoma).

All consecutive Patients who underwent successful PTMC for severe isolated Mitral stenosis and fulfilling the inclusion and exclusion criteria were included by non-probability consecutive technique after taking informed written consent. These patients were evaluated with transthoracic echocardiography (TTE) at echocardiography suite of National Institute of Cardiovascular Diseases Karachi before and 24 hours after PTMC. Their 12 leads ECG was also recorded. These patients were followed for six months and repeat TTE and 12 lead ECG was performed. Transesophageal echocardiography (TEE) was performed only in patients with atrial fibrillation to rule out left atrial/left atrial appendage thrombus.

PTMC was performed by double (Multitrack) double balloon technique in all cases. The procedure was performed by two experienced interventional cardiologist while monitoring the hemodynamics during the case.

All the patients with diabetes, hypertension, suboptimal PTMC, mitral regurgitation and aortic regurgitation more than grade 1, ECG or echocardiographic evidence of ischemia, Left Ventricle ejection fraction (LVEF) <50% and suspected peripartum cardiomyopathy were excluded from the study. The demographic, clinical and echocardiographic variables were entering through a specially designed proforma.

Transthoracic echocardiography was performed by a standard technique using Toshiba Xario 2100 echocardiographic machines. M-mode measurements were recorded according to American Society of Echocardiography criteria.⁵

Detailed 12 lead ECG was taken before, after 24 hours and 6 months after PTMC. It was read by two clinical cardiologists to see for P wave measurement in millisecond, QRS amplitude in V1 or V2 (the tallest of the two) in millivolt, Axis deviations (left, right or normal axis), quantification of axis on ECG (in degrees), bundle branch block i.e. either Right bundle branch block (complete/ incomplete) or Left bundle branch block, Biphasic P wave in Lead II, III, aVF (any of the three leads), ST depression in V1/V2 (greater of the two leads), R wave in V1 plus S wave in V5 in mV, axis of heart (degree).

All the data were analyzed by SPSS (Statistical Package for Social Sciences) Version 19.0 for Windows. Categorical variables were expressed as numbers and percentages while continuous variables like P wave width, QRS tallness, ST-T changes measurement and Right ventricular enlargement (R wave in V1 plus S wave in V5 in mV) were

expressed as mean±SD (Standard deviations). The McNemar test was used to analyze the study.

RESULTS

A total of 99 patients with severe isolated mitral stenosis who had undergone successful PTMC were studied. Females were 65(65.7%) and males were 34(34.3%). Mean age was 27.44 ± 6.26 years. Atrial fibrillation was documented in 25(25.3%) pre PTMC and 21(21.2%) post PTMC (p value 0.125). TTE performed before PTMC showed mean mitral valve area $0.89\text{cm} \pm 0.089\text{cm}^2$, mean LA diameter $4.65 \pm 0.82\text{cm}$ and mean right ventricular systolic pressure (RVSP) 62.3 ± 10.91 mm of Hg (Table 1). TTE 06 months after PTMC showed mean mitral valve area of $1.68 \pm 0.128\text{cm}^2$, mean mitral valve gradient 5.22 ± 1.21 mm of Hg, mean left atrial diameter 4.46 ± 0.65 cm and mean RVSP of 46.49 ± 7.8 mm of Hg (Table 2).

Standard 12 leads ECG Biphasic P wave was documented in 37(37.4%) pre PTMC and in 36(36.4%) post PTMC ($p < 0.01$). Pre PTMC complete/incomplete RBBB was present in 55 (55.5%) and LBBB in 1(1%) while post PTMC complete/incomplete RBBB was present in 54 (54.5%) and LBBB in 1(1%) (p 1.000) (Table 4). P wave amplitude was 0.26 ± 0.0491 mV pre PTMC and 0.24 ± 0.036 mV post PTMC ($p < 0.001$). P wave duration in V1/V2 (greater of the

two) was 114.32 ± 11.94 msec (80-140msec) and 108.64 ± 14.55 (80-140msec) pre and post PTMC respectively ($p < 0.001$). ST depression in V1/V2 (greater of the two) was $0.5758 \pm 0.55\text{mm}$ versus $0.5202 \pm .562\text{mm}$ pre and post PTMC ($p < 0.001$), R wave amplitude in V1/ V2 (greater of the two) $0.2879 \pm .2031$ mV vs. $0.2313 \pm 0.1475\text{mV}$ pre and post PTMC respectively ($p < 0.001$) as shown in table 3. Axis of the heart was 88.28 ± 19.51 degree (45-120) vs. 84.34 ± 19.79 degrees (30-120) pre and post PTMC respectively. RV hypertrophy as demonstrated by R wave in V1 plus S wave in V5 was 0.4830 ± 0.2239 millivolt vs. $0.3980 \pm .1823$ millivolt pre & post PTMC ($p < 0.001$) respectively. On average, pre PTMC P wave amplitude in V1/V2 value were 0.024 higher than post PTMC P wave amplitude in V1/V2 value (95% CI [0.016, 0.033]). There was a significant difference between pre and post RV end diastolic Diameter ($p < 0.001$). There was a significant difference between pre and post P wave duration in V1/V2 value ($p < 0.001$). On average, pre P wave duration in V1/V2 value were 5.68 higher than post P wave duration in V1/V2 value (95% CI [3.43, 7.91]). There was a significant difference between pre and post R wave in V1 plus S wave in V5 value ($p < 0.001$). On average, pre R wave in V1 plus S wave in V5 value were 0.086 higher than post R wave in V1 plus S wave in V5 value (95% CI [0.063, 0.109]).

Table 1: Demographic and Echocardiographic Characteristics of Study Population (n=99)

Characteristics	Numbers (n)	Percentage (%)
Age	27.44 ± 6.26 years	
Males	34	34.3%
Females	65	65.7%
Weight	48.4 ± 7.36 kg	
Height	156.7 ± 12.46 cm	
Spontaneous echo contrast	49	49.5%
Mitral valve area	$0.9\text{cm} \pm 0.089\text{cm}^2$	
LA diameter	4.65 ± 0.82 cm	
Mitral valve gradient	16.38 ± 2.51 mm of Hg	
Right ventricular diastolic diameter	22.59 ± 4.09 mm	
Right Ventricular systolic pressure	62.34 ± 10.98 mm of Hg	

Table 2: Echocardiographic Characteristics of Study Population {Post PTMC} (n=99)

Variables	Numbers (n)	Percentages (%)
Mitral valve area	$1.68 \pm 0.13\text{cm}^2$	
Spontaneous echo contrast	19	19.1%
LA diameter	4.46 ± 0.06 cm	
Mitral valve gradient	5.22 ± 1.21 mm of Hg	
Right ventricular diastolic diameter	20.1 ± 1.7 mm	
Right Ventricular systolic pressure	46.49 ± 7.83 mm of Hg	

Table 3: Quantitative ECG Variables of Study Population (n=99)

Variables		Numbers (n)	p-value
P wave amplitude	Pre PTMC (mV)	0.26 ± 0.0491	<0.001
	Post PTMC(mV)	0.24±0.036	
P wave width	Pre PTMC (m sec)	114.32± 11.94	<0.001
	Post PTMC(m sec)	108.64±14.55	
ST depression in V1/V2(greater among the two)	Pre PTMC(mm)	0.5758± 0.55	<0.001
	Post PTMC(mm)	0.5202±.562	
R wave amplitude in V1/V2 (greater of the two) in mV	Pre PTMC	0.2879±.2031	<0.001
	Post PTMC	0.2313±.1475	
Axis of the heart	Pre PTMC (Degree)	88.28±19.51	<0.001
	Post PTMC(Degree)	84.34±19.79	
R wave in V1 plus S wave in V5	Pre PTMC	0.4830± .2239	<0.001
	Post PTMC	0.3980±.1823	

Table 4: Qualitative ECG Variables of Study Population (n=99)

Variables		Numbers (n)	p-value
Complete/incomplete RBBB	Pre PTMC	55(55.5%)	1.00
	Post PTMC	54(54.5%)	
Right axis deviation	Pre PTMC	53(53.5%),	0.25
	Post PTMC	50(50.5%),	
Biphasic P wave	Pre PTMC	37(37.4%)	1.00
	Post PTMC	37(36.4%)	
Atrial fibrillation	Pre PTMC	25(25.2%)	0.125
	Post PTMC	21(21.2%)	

DISCUSSION

As evident from literature that after successful PTMC for severe isolated mitral stenosis there is regression of left atrial size, right ventricular pressure, right ventricular size, right atrial size, tricuspid regurgitation and pulmonary hypertension.¹⁻⁶ Our study shows very interesting findings. Qualitative data on Rhythm, Biphasic P wave, BBB and Axis of the Heart for pre and post PTMC shows no statistically significant difference, while quantitative variable i.e. magnitude of QRS complex in V1/V2 (longest of the two), P wave width, R wave in V1/V2 plus S wave in V5/V6 (showing right ventricular hypertrophy) showed that although there were subtle, but statistically significant changes. In our study population there was no evidence of pulmonary pathologies leading to secondary right ventricular failure as the exclusion criteria were strictly followed. There was a significant difference between pre and post PTMC P wave amplitude in V1/V2 value ($p < 0.001$). On average, pre PTMC P wave amplitude in V1/V2 value were 0.024 higher than post PTMC P wave amplitude in V1/V2 value (95% CI [0.016-0.033]). There was a significant difference between pre and post P

wave duration in V1/V2 value ($t_{73} = 5.05$, $p < 0.001$). On average, pre P wave duration in V1/V2 value were 5.68 higher than post P wave duration in V1/V2 value (95% CI [3.43, 7.91]). There was a significant difference between pre and post PTMC R wave in V1 plus S wave in V5 value ($t_{98} = 7.474$, $p < 0.001$). In a study by Hamdy up to 61.54% of patients normalized their RV systolic function after successful PTMC.⁶ The rest of patients may have either static RV dysfunction or having progressive decline.⁶ In the same direction of our results, Mohan, et al. found that, in spite of the significant drop of pulmonary pressure after successful valvuloplasty, the immediate improvement in RV systolic function was not clear, and about 65% of patients improved within one year duration and their RV systolic function returned to normal values.^{7,8} Right ventricular (RV) function plays an important role in the development of clinical symptoms, exercise capacity, prognosis, and survival in patients having MS.⁹⁻¹³ Similar to these aforementioned studies it is obvious from our echocardiographic findings in this study that RV function significantly improves after successful PTMC i.e. RVSP decreased from 62 to about 42 mm of Hg. Similarly there was a significant changes in the

ECG variables that directly measures the RV pressure i.e. R wave in V1/V2 plus S wave in V5/V6 and the magnitude of QRS complex in V1/V2 (longest of the two). On average, pre R wave in V1 plus S wave in V5 value were 0.086 higher than post R wave in V1 plus S wave in V5 value (95% CI [0.063, 0.109]) in our study.

There is scant data in international literature regarding the ECG changes associated with successful PTMC, but all the data present on this topic support our findings. In a study published in 1968 showed that there is subjective improvement in some of ECG variables for the morphological changes in dimensions of the heart. The mean age was slight more than our study i.e. 37 years.¹⁴ They analyzed 12-lead ECGs before and after surgery. The criterion of P wave 0.12 sec. or more demonstrated the greatest postoperative decrease (26%). Group A (improvement group) demonstrated a greater postoperative normalization of all the P wave parameters than group B (no improvement) in their study. Significant changes in group A consisted of a decrease in the P duration from 0.12 to 0.10 sec. ($p < 0.001$) and P amplitude from 0.18 to 0.14 mv ($p < 0.001$). Decreases were smaller and not statistically significant in group B. Their findings conclude that a decrease in P wave amplitude of 0.05 mv or more and P wave duration of 0.02 sec. or more are associated with objective clinical improvement after open heart mitral commissurotomy. This study support our findings that there was a quantitative improvement of P wave dimension of about similar valves in our study.¹⁴ In another study by Ragab et al which is published in 2013 showed that P-wave maximum and dispersion are significantly increased in patients with mitral stenosis. These changes decreased significantly after PTMC. It was concluded from their study that the degree of and the changes in P-maximum and P-wave dispersion were correlated with development of late cardiac events after PTMC, with Cut-off values of ≥ 62.8 millisecond for P-wave dispersion and 118 millisecond for P-maximum. So it support our findings of successful PTMC have decrement in the P wave.¹⁵ Similar to our study B1 Chandrasekar et al. studied the effect of acute hemodynamic changes that occur following PTMC on the ECG in 25 patients with isolated rheumatic mitral stenosis. They observed significant changes in the characteristics of P-wave and QRS axis. Patients who showed changes in P-wave had significantly greater fall in left atrial mean pressure ($p < 0.025$). Good correlation was seen between the decrease in right axis deviation of QRS axis and fall in mean pulmonary artery pressure ($r = +0.56$, $p < 0.001$) and pulmonary vascular resistance ($r = +0.48$, $p < 0.05$). All these ECG changes was demonstrated within 72 hours in their study. These results demonstrate that the acute hemodynamic changes following balloon mitral valvotomy produce corresponding changes on electrocardiogram and these changes indicate a significantly greater degree of hemodynamic benefit from

the procedure than when these changes are not seen.¹⁶ Similarly ET José Carlos et al found that P-wave duration decreased from 0.12 ± 0.01 sec pre-PMV to 0.09 ± 0.02 sec at 2 months follow-up (< 0.0001) was the only independent predictor of decrease left atrial size and so of the future events after PMV.¹⁷ The only limitation of the translation of the ECG changes with successful PTMC is the presence of atrial fibrillation. As AF is not frequently reverted to persistent normal sinus rhythm after successful PTMC as evident from our study, so in atrial fibrillation left atrial size changes after successful PTMC are not translated to ECG as there is no definite P wave. As Atrial fibrillation was documented in 25 patients' pre PTMC and 21 patients post PTMC, so the P wave was analyzed only in 74 patient's pre PTMC and 78 patients post PTMC. ECG changes may be useful in prompting further diagnostic evaluation for patients with mitral stenosis having successful PTMC in follow up. Although echocardiography is a non-invasive and reproducible method to evaluate cardiac functions, it should be kept in mind that RV function parameters are also not fully independent parameters. ECG changes if studied in randomized control trails can be a useful tool to see for mid to long term follow up of these patients especially in the developing countries where echocardiography is not readily available especially for right ventricular functional assessment which is technically demanding. Our observational study provides ground for further research on this subject especially in developing countries where rheumatic heart disease is still endemic. To the best of our knowledge this is the first comprehensive study of its kind which directly focuses on the ECG changes after successful PTMC. This study will enlightens way to further research on subtle ECG changes after PTMC.

CONCLUSION

ECG changes are directly proportional to the echocardiographic improvement in cardiac chambers i.e. the morphological changes in the heart after successful PTMC in isolated severe mitral stenosis patients.

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