The Role of Three Dimensional Transesophageal Echocardiography for Assessment of Tricuspid Regurgitation after Percutaneous Balloon Mitral Valvuloplasty

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Abstract: **Background:** Tricuspid regurgitation (TR) is an important predictor of morbidity and mortality in rheumatic mitral stenosis (MS) and in the majority of cases it is functional. **Introduction:** It is unclear whether mitral valve (MV) surgery with simultaneous surgical correction of TR would be the better treatment option in patients who are potential candidates for percutaneous mitral balloon valvuloplasty (PMBV), because MV surgery has the morbidity and mortality related with operation, and regression of TR may result from improvement of pulmonary hypertension after PMBV. **Aim of the work:** To investigate the effect of PMBV on the tricuspid annulus (TA) and tricuspid regurgitation by three dimensional transesophageal echocardiography (3D TEE). **Material and methods:** Twenty five patients (M/F=5/20) with rheumatic MS referred for PMBV between October 2014 and October 2016 were included in our study. 3D TEE was done pre PMBV and 48hr post PMBV. For tricuspid valve (TV) analysis, we adapted commercially available software designed for assessing the mitral valve (MVQ software, Philips). TA diameters and area (TAA) were measured at end diastole & end systole at 2 cycles taking their average. TR was assessed by vena contracta area (VC area) measured at mid-systole offline using Philips Qlab. **Results**: PMBV was successfully completed in all 25 patients with MR post BMV not more than grade II/IV. MVA_{3D} increased significantly post PMBV & was significantly correlated with MVA_{2D} (P value <0.0001). MS increases the RV afterload which causes stress stretching effect over the tricuspid annulus causing it to be dilated, circular and planer. Post PMBV, TA regress in size with better function and starts the way to regain some of its normal geometrical oval saddle shape appearance. PMBV results in alleviating tricuspid regurgitation severity as assessed by TR VC area (P-value = 0.0017). **Conclusion:** 3D TEE allowed us to examine the TV in great detail**.** We concluded that tricuspid annulus dimensions and function start to improve and TR starts to regress after PMBV. **Recommendations:** 3D TEE can be a great tool for a comprehensive assessment of morphology and function of the TV complex. Also, our study supports early intervention in MS patients once indicated prior to the development of significant pulmonary hypertension, right ventricle myocardial dysfunction or significant functional TR.
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Keywords: Tricuspid regurgitation – Mitral valvuloplasty - 3D echocardiography

1. Introduction:

Rheumatic mitral stenosis still remains an important public health concern particularly in developing countries. Mitral stenosis is occasionally associated with tricuspid regurgitation (TR) of variable grades and in the majority of cases it is functional, resulting from right ventricular and tricuspid annular dilation caused by chronic pulmonary hypertension and this association has an adverse impact on morbidity and mortality **(Pastore S, et al., 2011).**

However, it is unclear whether MV surgery with simultaneous surgical correction of TR would be the better treatment option in patients who are potential candidates for PMBV, because MV surgery has the morbidity and mortality related with operation, and regression of TR may result from improvement of pulmonary hypertension by PMBV **(Song JM, et al., 2003).**

Percutaneous mitral balloon valvuloplasty (PMBV) is a common treatment used in patients with mitral stenosis. The effect of successful PMBV on the right heart in patients with rheumatic MS has not been well defined **(Sanchez, et al., 2013).**

Three-dimensional echocardiography is a new modality, which can overcome geometric complexity of the RV & tricuspid valve to some extent. Three-dimensional echocardiography allows users to visualize the TV apparatus from any perspective. This may significantly improve our understanding of the pathophysiological mechanisms underlying the various TV diseases and functional tricuspid regurgitation, and potentially suggest ways to improve surgical treatment **(Kjaergaard J, et al., 2006).**

Previous reports found good relationships between the area of the vena contracta measured with 3D Echocardiography and conventional estimates of tricuspid regurgitation severity by 2D Echocardiography color Doppler **(Velayudhan, et al., 2006).**

As our knowledge on three dimensional echocardiography is still growing, we sought to investigate the effect of percutaneous balloon mitral valvuloplasty on tricuspid annulus & tricuspid regurgitation by three dimensional transesophageal echocardiography.

2. Material and methods:

Twenty five patients (M/F=5/20) with rheumatic MS referred to cardiology department, Bab Alsharyia hospital, Al Azhar university for PMBV between October 2014 and October 2016 were included in our study. Patients were subjected to full history taking, clinical evaluation, ECG, conventional 2D, M mode, Doppler echocardiography. 3D TEE was done pre PMBV and 48hr post PMBV. PMBV was successfully completed in all 25 patients with MR post BMV not more than grade II/IV. For tricuspid valve (TV) analysis, we adapted commercially available software designed for assessing the mitral valve (MVQ software, Philips). TA diameters and area (TAA) were measured at end diastole & end systole at 2 cycles taking their average. TR was assessed by vena contracta area measured at mid-systole offline using Philips Qlab. All data were collected and statistically analyzed using the statistical version of the Graph Pad InStat.

3. Results:

Our study included 25 patient, 5 males (20%) and 20 females (80%). Mean age ± SD was (31.2 ± 5.32) years. Mean BMI ± SD was (23.75 ± 1.708) kg⁄m².

The conventional transthoracic echocardiography data of all patients are listed in table (1).

Table (1): Shows transthoracic echocardiography (TTE) data of all patients:

TTE data	***Pre PMBV (Mean± SD)***	***Post PMBV (Mean± SD)***	***P value***
LVEDD (cm)	4.9 ± 0.57	4.8 ± 0.53	0.448
LVESD (cm)	3.1 ± 0.51	2.9 ± 0.49	0.4205
LV EF %	64.78 ± 6.5	66.24 ± 6.19	0.4205
MVA_{2D} (cm^2)	0.98 ± 0.155	1.95 ± 0.22	< 0.0001*
MVA_{PHT} (cm^2)	0.91 ± 0.166	1.78 ± 0.217	< 0.0001*
Transmitral MPG(mmHg)	17.52 ± 5.99	5.56 ± 1.26	< 0.0001*
LAD (cm)	5.1 ± 0.56	4.76 ± 0.54	0.0362*
RVD (cm)	3.46 ± 0.38	3.24 ± 0.37	0.0466*
RAA (cm^2)	13.7 ± 3.4	12.8 ± 3.4	0.3864
RV EF %	46.008 ± 9.70	51.79 ± 8.26	0.0277*
RVFAC %	35.512 ± 6.28	40.64 ± 7.15	0.0097*
RV Tei index	0.55 ± 0.084	0.49 ± 0.065	0.0048*
PASP (mmHg)	65.64 ± 18.905	41.28 ± 8.005	< 0.0001*
PVR (WU)	2.91 ± 1.93	1.81 ± 1.004	0.0150*
TR venacontracta (mm)	4.82 ± 1.285	3.77 ± 1.045	0.0027*
TRJA/RAA ratio	0.33 ± 0.099	0.25 ± 0.07	0.0034*

LVEDD: *Left ventricle end diastolic dimension* ***LVESD:*** *Left ventricle end systolic dimension* ***LVEF:*** *Left ventricle ejection fraction* ***MVA_{2D}:*** *mitral valve area measured by 2D planimetry* ***MVA_{PHT}:*** *mitral valve area measured by pressure half time* ***MPG:*** *mean pressure gradient* ***LAD****: left atrial diameter* ***RVD****: Right ventricle diameter* ***RAA:*** *Right atrial area* ***RVEF****: Right ventricle ejection fraction* ***RVFAC:*** *Right ventricle fractional area change* ***PASP****: Pulmonary artery systolic pressure* ***PVR****: Pulmonary vascular resistance* ***TRJA:*** *Tricuspid regurgitation jet area* ***WU:*** *Wood unit*

PMBV was successfully completed in all 25 patients with MR post BMV not more than grade II/IV. There was significant increase in mitral valve area measured by 2D planimetry method (MVA_{2D}) and pressure half time method (MVA_{PHT}) (P value < 0.0001). PMBV results in significant transmitral mean pressure gradient (MPG) decrease (P value < 0.0001) & this was correlated with catheterization lab measurements of left atrial pressure & pressure gradient across the mitral valve, as there was significant reduction in left atrial pressure(P value < 0.0001) & Pressure gradient across MV (P value < 0.0001) Post PMBV.

No significant changes in vital signs as heart rate, systolic blood pressure or diastolic blood pressure occurred post PMBV. Also, No significant changes in LV end diastolic dimension (P value = 0.448) or LV end systolic dimension (P value = 0.4205) occurred Post PMBV. Also, No significant changes in LV EF (P value = 0.4205). Left atrial diameter (LAD) shows significant decrease after PMBV (P =0.0362). Right ventricle diameter (RVD) shows significant decrease after PMBV (P =0.0466). Right atrial area (RAA) shows no significant decrease after PMBV (P =0.3864).

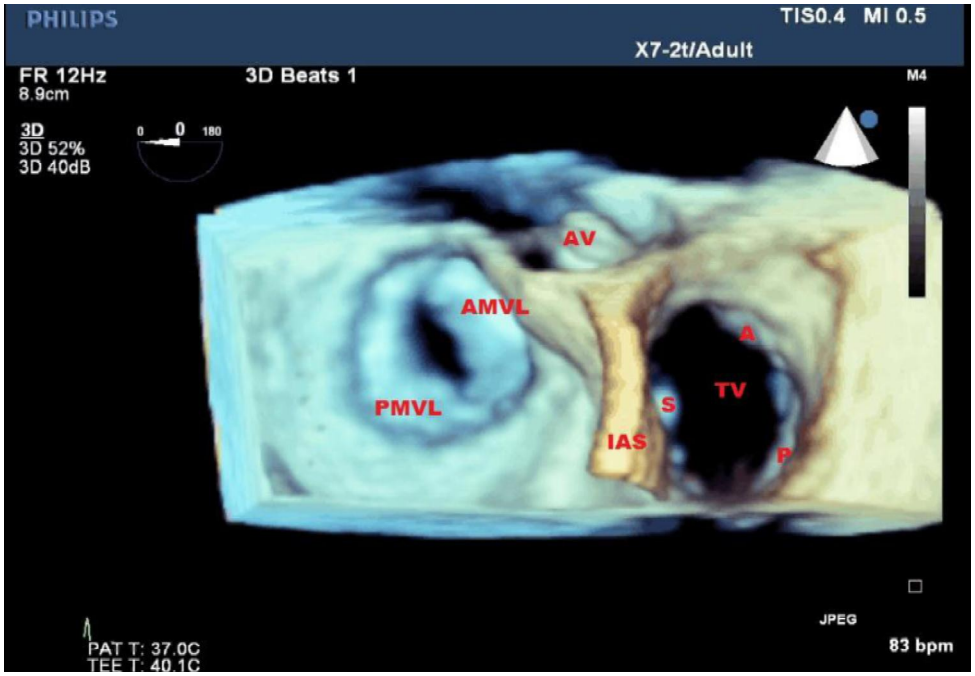

Figure (1) 3D TEE, 3D Zoom mode, atrial aspect demonstrated the mitral stenosis Pre PMBV & tricuspid valve leaflets while opened during diastole.
*(**AMVL**: Anterior mitral valve leaflet **PMVL**: posterior mitral valve leaflet **AV**: Aortic valve **IAS**: Interatrial septum **TV:** tricuspid valve **S:** Septal leaflet of TV **A**: Anterior leaflet of TV **P**: Posterior leaflet of TV)*

Right ventricle ejection fraction (RVEF) and fractional area change (RVFAC) show significant increase after PMBV (P=0.0277, P= 0.0097 respectively). There was statistically significant decrease in Myocardial Performance Index (Tei index) post PMBV (P-value =0.0048). The pulmonary artery systolic pressure (PASP) & pulmonary vascular resistance (PVR) decreased significantly Post PMBV (P <0.0001, P=0.0150 respectively).

There was significant regression in tricuspid regurgitation severity as evidenced by decrease in TR vena contracta (P = 0.0027) & TR jet area /RA area ratio (P = 0.0034).

The 3D TEE data of all patients are listed in table (2). In our study, MVA_{3D} (Mitral valve area measured by 3D TEE) increased significantly post PMBV & was significantly correlated with MVA_{2D} (Mitral valve area measured by 2D planimetry method), (P value <0.0001). *Figure 2 & 3.*

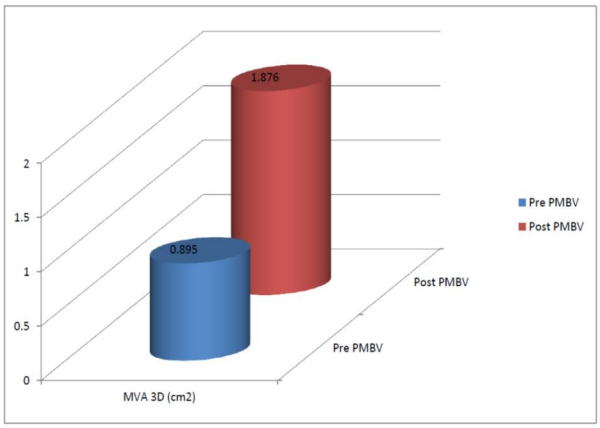

Figure (2) comparison between pre PMBV & post PMBV MVA $_{3D}$ (cm^2)

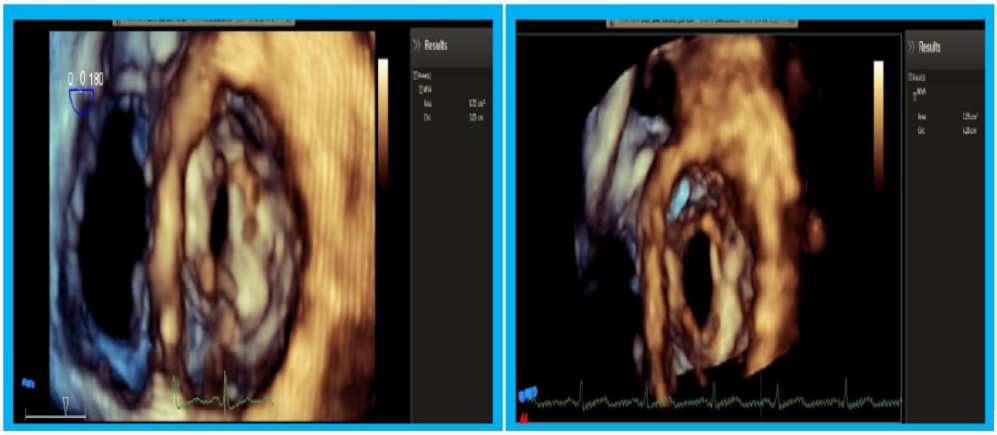

Figure (3) 3D TEE, 3D Zoom mode from ventricular aspect showin MVA pre PMBV (Left), MVA Post PMBV (Right).

The tricuspid annulus late diastolic anteroposterior diameter (TADAP), TA late diastolic septolateral diameter (TADSL), TA late systolic anteroposterior diameter (TASAP) & TA late systolic septolateral diameter (TASSL) decreased significantly post PMBV (P = 0.0486, 0.0482, 0.0451 & 0.0005 respectively). Also, both late diastolic TA area (TAA_D) & late systolic TA area (TAA_S) decreased significantly post PMBV (P = 0.0479 & 0.0211 respectively). *Figure 4.*

There was significant negative correlation between MVA_{3D} & TAA_D with P value = 0.0337 (The increase in MVA Post PMBV was associated with decrease in TA late diastolic Area). There was significant negative correlation between MVA_{3D} & TAA_S with P value = 0.0195 (The increase in MVA Post PMBV was associated with decrease in TA systolic area). There was significant correlation between TAA & pulmonary artery pressure with P value = 0.0003 (TA dilates with pulmonary hypertension & decreases in size with relieve of this pulmonary hypertension).

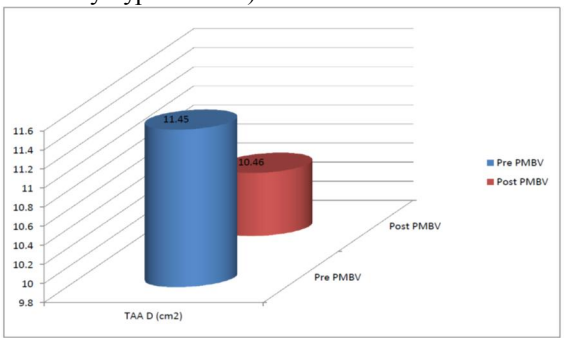

Figure (4) comparison between pre PMBV & post PMBV TAA_D (cm^2).

Table (2): Shows three dimensional transeosophageal echocardiography (3D TEE) data of all patients:

3D TEE data	***Pre PMBV (Mean± SD)***	***Post PMBV (Mean± SD)***	***P value***
MVA_{3D} (cm^2)	0.895 ± 0.149	1.876 ± 0.194	< 0.0001*
TADAP (cm)	3.6 ± 0.35	3.4 ± 0.32	0.0486*
TASAP (cm)	3.4 ± 0.37	3.2 ± 0.35	0.0451*
TADSL (cm)	3.5 ± 0.39	3.3 ± 0.32	0.0482*
TASSL (cm)	3.1 ± 0.38	2.7 ± 0.34	0.0005*
TAA_D (cm^2)	11.45 ± 1.87	10.46 ± 1.53	0.0479*
TAA_S (cm^2)	9.098 ± 1.65	8.094 ± 1.30	0.0211*
TAFAC %	20.54 ± 3.624	22.66 ± 3.625	0.0437*
TR Vena Contracta Area (cm^2)	0.58 ± 0.120	0.46 ± 0.129	0.0017*
TA Eccentricity Ratio	1.117 ± 0.127	1.193 ± 0.122	0.0380*
TA Height (mm)	4.048 ± 1.048	4.724 ± 0.994	0.0235*
TA Stretch Ratio	9.26 ± 3.56	7.14 ± 1.91	0.0116*
TV Tenting Volume (ml)	1.46 ± 0.78	0.99 ± 0.53	0.0177*

MVA_{3D}: *mitral valve area measured by 3D TEE* ***TADAP:*** *tricuspid annular diastolic anteroposterior diameter* ***TASAP:*** *tricuspid annular systolic anteroposterior diameter* ***TADSL:*** *tricuspid annular diastolic septolateral diameter* ***TASSL:*** *tricuspid annular systolic septolateral diameter* ***TAA_D:*** *Diastolic tricuspid annular area* ***TAA_S:*** *Systolic tricuspid annular area* ***TAFAC:*** *Tricuspid annular fractional area change* ***TA****: Tricuspid annulus*

TA height was increased post PMBV (P-value = 0.0235). Also, TA stretch ratio(TASAP/TA Height) decreased post PMBV (P-value = 0.0116). This means that the tricuspid annulus as reported by previous studies has saddle shape with high points at the anteroposterior axis towards the right atrium and low points at the septolateral axis towards the right ventricle. Also, in our study TA eccentricity ratio was calculated from TASAP/TASSL ratio which can assess circularity of TA, with increased circularity as this ratio approaches (1). It was 1.117 ± 0.127 pre PMBV & increased to 1.193 ± 0.122 post PMBV (P value = 0.0380). It seems that in MS, the high RV after load and pulmonary hypertension tend to have stress stretching effect over the TA making it tend to be more planer (less saddle shape) & more circular (less oval). Post PMBV and with acute decrease in RV after load, TA tend to regain its normal shape and become less planer (more towards its normal Saddle shape appearance) and less circular (more towards its normal oval shape appearance). *Figure 5.*

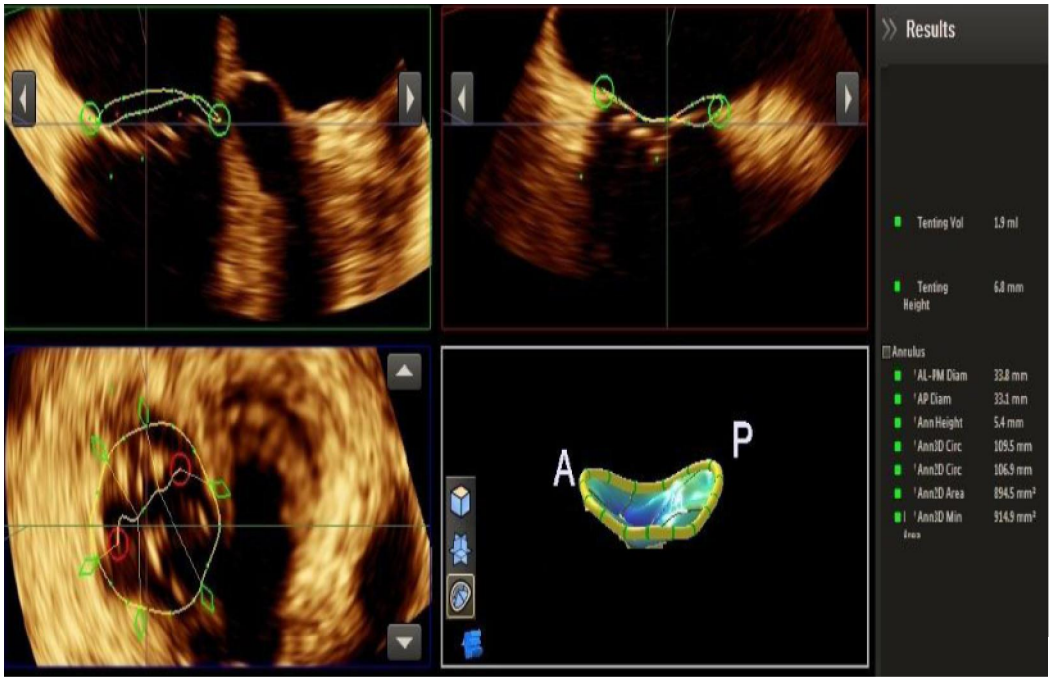

Figure (5) 3D TEE pre PMBV, Tricuspid annular model & measurements (TASAP, TASSL, TAA $_S$, TA Height, TV tenting volume & Height) by offline analysis using Philips Qlab, adapting MV quantification software.

In our study, we used Tricuspid Annular Fractional Area change (TAFAC) calculated from: ((Diastolic TAA – Systolic TAA) / Diastolic TAA) as a marker for recovery of the RV function post PMBV in MS patients. There was statistically significant increase in TAFAC % Post PMBV (P-value = 0.0437). There was significant positive correlation between MVA_{3D} & TAFAC % with P value = 0.0482(which means that the increase in MVA Post PMBV was associated with better TA function). Also, there was significant positive correlation between TAFAC % measured by RT- 3D TEE & RVFAC % measured by 2D TTE with P value = 0.0412 (which means that TAFAC % can be good marker of RV function).

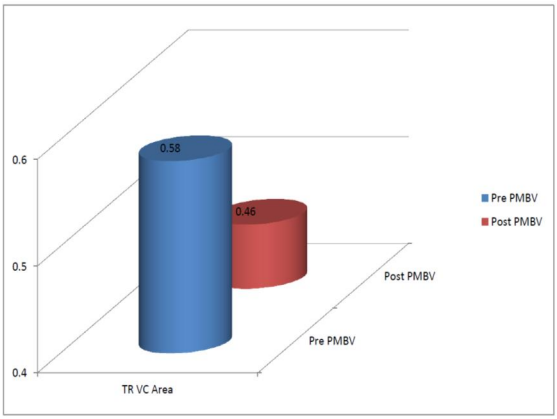

Figure (6) Comparison between pre PMBV & post PMBV TR VC area (cm^2).

As regard to the effect of PMBV on the TR severity, we used TR VC area measured by RT-3D TEE for TR evaluation. There was statistically significant decrease in TR VC area post PMBV (P-value = 0.0017), figure 6. There was significant negative correlation between MVA_{3D} & TR VC area (P value = 0.0030) which means that increasing MVA post PMBV has good impact on TR severity regression.

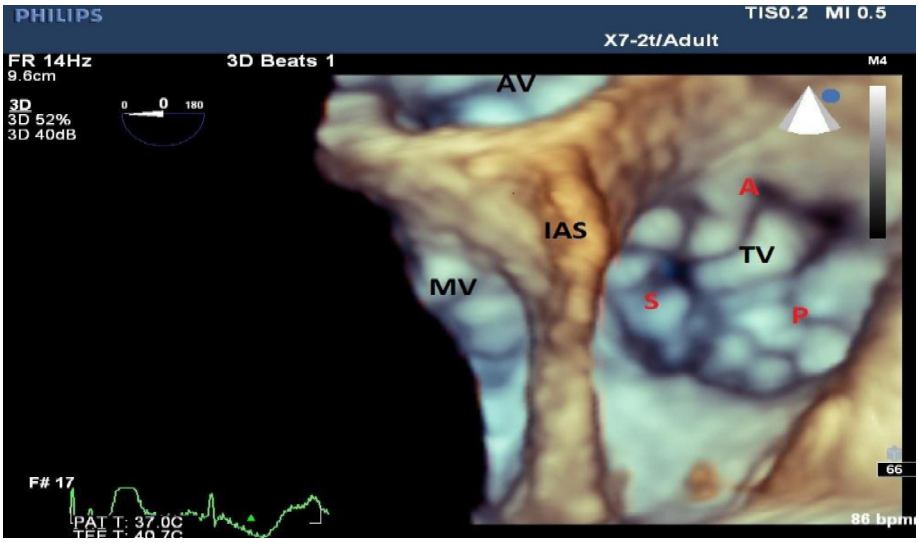

Figure (7) 3D TEE pre PMBV, 3D Zoom mode clearly demonstrating the tricuspid valve leaflets & the regurgitant orifice.
AV: Aortic valve ***IAS****: Interatrial septum* ***TV:*** *tricuspid valve* ***MV:*** *mitral valve* ***S:*** *Septal leaflet of TV* ***A****: Anterior leaflet of TV* ***P****: Posterior leaflet of TV.*

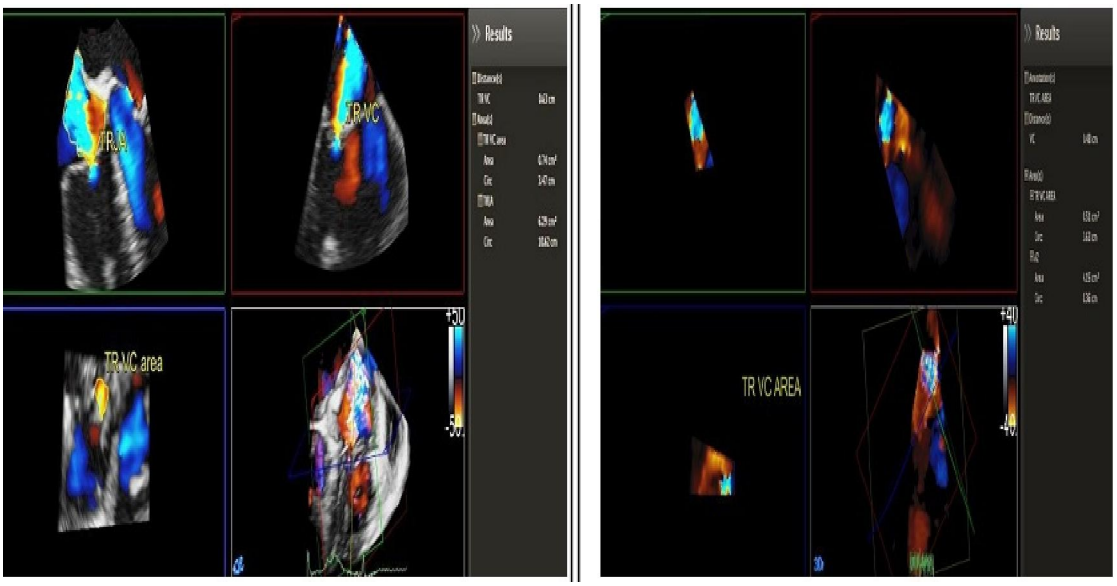

Figure (8) TR VC area measured by 3D TEE offline analysis using Philips Qlab, 3DQ software, Pre PMBV (Left) & Post PMBV (Right)

We have found that many factors play an important role in the pathophysiology of the functional TR in MS and they are related to TR severity regression post PMBV. These factors are pulmonary artery pressure, tricuspid annular area specifically systolic TAA (TAA_S), tricuspid annular function (TAFAC%) & TV tenting Volume. There was significant correlation between pulmonary artery pressure & TR VC area with P value = 0.0005(which means that pulmonary artery pressure decrease post PMBV cause regression in TR severity), *Figure 11*. Also, there was significant correlation between TAA_S

& TR VC area with P value < 0.0001 (which means that tricuspid annular area regression post PMBV results in alleviating TR severity post PMBV), *Figure 9*. Also, There was significant negative correlation between TAFAC % & TR VC area with P value = 0.0010 (which means that TR regress with TA function improvement). Also, There was statistically significant decrease in TV tenting volume measured at mid systole post PMBV (P-value = 0.0177) & There was significant correlation between TV Tenting Volume & TR VC area (with P value = 0.0011) which means that TV tenting volume was an important factor affecting TR severity & the decrease in TV tenting volume Post PMBV can explain TR regression post PMBV), *Figure 10*.

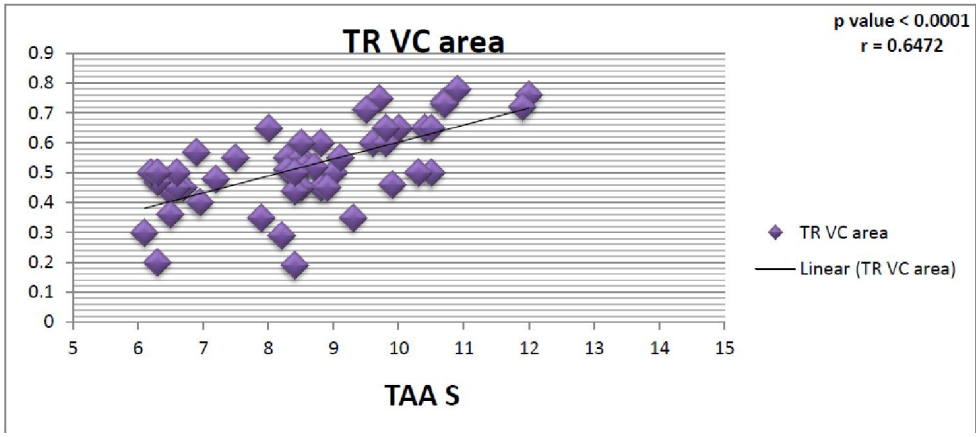

Figure (9) Correlation between TAA $_S$ & TR VC area

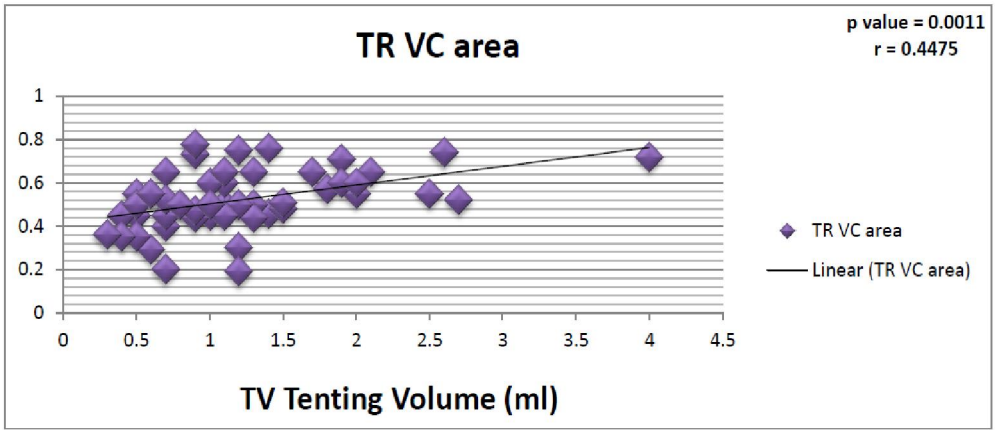

Figure (10) Correlation between TV tenting volume & TR VC area.

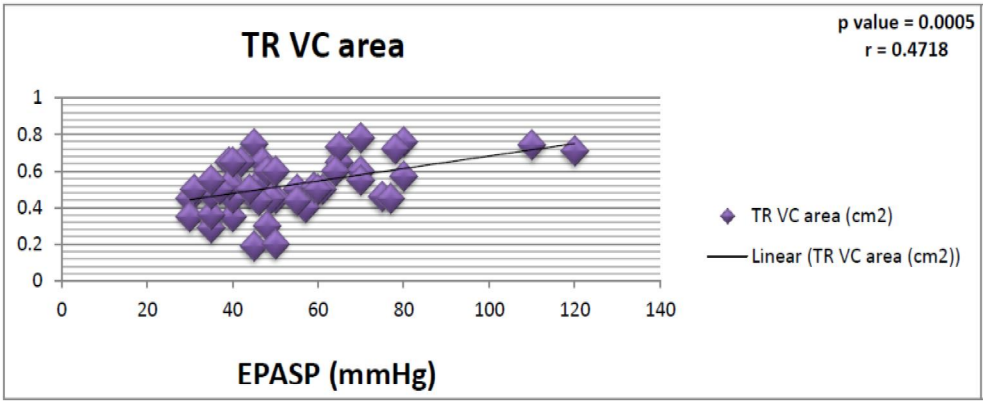

Figure (11) Correlation between PASP & TR VC area.

4. Discussion:

Tricuspid valve function plays an important role in mitral stenosis and the development of functional tricuspid regurgitation is directly associated with increased morbidity and mortality **(Nath J, et al., 2004)**.

The transthoracic echocardiography findings of this study support previous reports which suggest that PMBV results in recovery of the right ventricle function (a decrease in RV Tei index, and pulmonary arterial pressure, Pulmonary Vascular Resistance, and an increase in RVEF & RVFAC) which can be explained by the recovery of the RV outflow tract systolic functions due to the acute decrease in the RV afterload. Also, these results demonstrated the effect of PMBV on alleviating the severity of TR **(S Ghaffari, et al., 2009) and (İnci, et al., 2014).**

In our study, MVA_{3D} increased significantly post PMBV & was significantly correlated with MVA_{2D} and this was in agreement with other previous studies **(Anwar, et al., 2010) and (Dreyfus J, et al.,2011).**

As regard TA dimensions results of our study, we suggest that MS results in progressing pulmonary hypertension & increasing RV afterload which causes stress effect over the tricuspid annulus causing it to be dilated, tend to be circular (less oval) & tend to be planer (less saddle). Post PMBV, TA dilation starts the way to be partially relieved & TA starts the way to regain some of its normal oval saddle shape appearance mostly due to the recovery of the RV outflow tract systolic functions due to the acute decrease in the RV afterload. These results were in agreement with many previous studies. **(Liam Ring, et al., 2012), (Dreyfus, et al., 2015) and (Ton-Nu, et al., 2006).**

In our study, PMBV results in improvement of the tricuspid annular function as assessed by TAFAC% measured by 3D TEE and this may suggest recovery of the RV function post PMBV. This is in agreement with previous study in which the morphological and functional aspects of the TA were assessed by 3D TTE **(Anwar, et al., 2007).**

PMBV results in alleviating tricuspid regurgitation severity as assessed by TR VC area measured by 3D TEE. This is due to decrease of pulmonary artery Pressure, improvement of the tricuspid annular function, decrease of the tricuspid annular dilation & flattening (i.e. stretching) and decrease of the TV tenting volume.

Previous Reports demonstrated that 3D echocardiography can help in estimating the severity of TV regurgitation using color flow. They demonstrated the feasibility of obtaining the area of the vena contracta of the tricuspid regurgitant jet by cropping the 3D echocardiography color Doppler data set **(Velayudhan, et al., 2006)**.

Ton-Nu et al, in 2006 examined the 3D geometry of the tricuspid valve annulus (TVA) in patients with functional TR, comparing them with patients with normal tricuspid valve function and relating annular geometric changes to functional TR. With significant functional TR, the annulus becomes larger, more planer & circular & this in agreement with our study.

Miglioranza et al. in 2014 evaluated the role of 3D echocardiography measured TA surface area (TASA) to predict the severity of functional tricuspid regurgitation (FTR). They reported that TASA has a greater predictive power to discriminate severe from non-severe FTR.

Spinner EM et al, in 2012 concluded that alterations in ventricular geometry can lead to TR by altering both tricuspid annulus size and papillary muscle position & this is in agreement with our study.

Hiroki Ikenaga, et al., in 2014 suggested a possible association between TA dilation and the TR severity & this is in agreement with our study.

Conclusion:

3D TEE allows us to examine the TV in great detail**.** We concluded that after PMBV, TR severity starts to regress. Also, TA starts the way to regain some of its normal geometrical oval saddle shape appearance and its function starts to improve. This may indicate the importance of intervention in MS patients once indicated prior to the development of significant pulmonary hypertension, right ventricle myocardial dysfunction or significant functional TR.

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