Mitral kapak replasmanı sonrasında pulmoner fonksiyonlarda uzun dönem takipte ortaya çıkan değişiklikler

Alterations of pulmonary functions occuring after mitral valve replacement in long-term follow-up

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Özet

Amaç: *Bu çalışmada, mitral kapak hastalığı nedeni ile mitral kapak replasmanı uygulanmış hastalarda pulmoner fonksiyonlardaki değişikliklerin geri dönüşümlü olup olmadığını değerlendirmeyi amaçladık.*

Gereç ve yöntem: *Kliniğimizde Ocak 2008 ile Haziran 2011 tarihleri arasında mitral kapak replasmanı uyguladığımız 26 hasta değerlendirildi. Aort yetersizliği, koroner arter hastalığı, kronik obstrüktif akciğer hastalığı olan hastalar çalışmaya alınmadı. Hastaların demografik özellikleri, ekokardiyografi bulguları, spirometrik solunum fonksiyon test sonuçları kaydedildi. İstatistiksel analiz için SPSS for Windows 10.0 (statistical package for social sciences) programı kullanıldı.*

Bulgular: *Çalışmaya alınan hastalar retrospektif olarak değerlendirildi. FVC, FEV1, FEV1/FVC, FEF 25-75 %, PEF değerleri pre ve postoperatif olarak karşılaştırıldı. p<0,001 değerini istatistiksel olarak anlamlı kabul ettik. Değerlendirmeye aldığımız tüm veriler istatistiksel olarak anlamlı idi.*

Sonuç: *Mitral kapak hastalığına bağlı olarak ortaya çıkan pulmoner fonksiyonlardaki bozulmanın önemli bir kısmının geri dönüşlü olduğunu düşünmekteyiz.*

Anahtar kelimeler: *mitral kapak, hastalık, hipertansiyon, solunum, fonksiyon testi*

Türkçe kısa makale başlığı: *MVR sonrasında pulmoner fizyodinami*

Abstract

Objective: *In this study, we aimed to evaluate the reversibility of the alterations of pulmonary functions in the group of patients who underwent mitral valve surgery due to mitral valve disease.*

Methods: *In this study, 26 of patients were evaluated who underwent mitral valve replacement in our clinic between January 2008 and June 2011. The patients with aortic regurgitation, coronary artery disease, chronical obstructive lung disease were excluded. Patients' demographic characteristics, echocardiographical findings and spirometrical pulmonary function test results were recorded. SPSS (statistical package for social sciences) for Windows 10.0 programme was used for statistical analysis.*

Results: *Study group of patients were evaluated retrospectively. FVC, FEV1, FEV1/FVC, FEF 25-75 %, PEF values were compared pre and postoperatively. We considered p<0,001 as statistically significant. All measures we evaluated were statistically significant.*

Conclusion: *We concluded that the important part of the pulmonary dysfunctions due to mitral valve disease are reversible.*

Key words: *mitral valve, disease, hypertension, pulmonary, function test*

İngilizce kısa makale başlığı: *Pulmonary physiodinamics after MVR*

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Introduction

Physiology of the mitral valve diseases cause alterations in the pulmonary vascular bed. Pulmonary arterial pressures increase in resting time in patients with chronic severe mitral stenosis in which the pulmonary vascular resistance developed.

When the systolic pressures of pulmonary artery become higher than 60 mmHg, higher resistance exists over the right ventricle. When the end-diastolic pressures of right ventricle increase right atrial pressures also increase at the same time. Pulmonary arteriolar vasoconstruction due to left atrial hypertension causes increase of the pulmonary vuscular resistance (1,2).

Passive transmission of high pressure of left atria causes pulmonary venous hypertension, pulmonary arteriolar construction, obstructive alterations of pulmonary vascular bed and pulmonary hypertension respectively. Progressive increase of pulmonary hypertension leads to right cardiac failure, tricuspid valve regurgitation, and pulmonary valve regurgitation (2,3).

Severe mitral stenosis leads to irreversable pulmonary vascular alterations in long term period.[4] Because of higher occurance of chronically increased pressure of left atria in mitral stenosis, pulmonary vascular resistance of the patients with mitral stenosis is higher than the patients with mitral regurgitation. Abrogating the mitral valve pathology by mitral valve replacement leads to intracardiac pressures decrease to normal levels and improvement of pulmonary functions. Nevertheless some of the patients' cardiac pressures do not improve and the pulmonary dysfunction persists. In the cases with irreversible pulmonary dysfunction, one of the determining factors of mortality and morbidity is passive pulmonary congestion that exists in operative period.

Cardiopulmonary bypass induces systemic inflammatory response and leads to capillary leakage syndrome which causes edema in all tissues and end-organ dysfunction at the healing period. Pulmonary dysfunction is one of the most frequent organ dysfunction (5,6)

In this study, we aimed to evaluate the reversability of the restructive and obstructive pulmonary changes in the group of patients with mitral valve disease who underwent mitral valve surgery.

Methods

In this study, 26 patients were evaluated who underwent mitral valve replacement between January 2008 and June 2011 retrospectively. This study was approved by the ethical committee. The patients with aortic regurgitation, coronary artery disease, chronic obstructive pulmonary disease were excluded. The demographical characteristics such as age, gender, lenght, weight, body surface area, transthoracic echocardiographic findings, pulmonary function tests were recorded. The pulmonary functions were evaluated by using spirometry (ZAN600 Ergospirometry nSpire Health Schlimpfhofer Strasse 14D97723 Oberthulba) three days before and six months after the operation respectively.

Forced Vital Capacity (FVC), Forced Expression Volume in first second (FEV1), Maximum Flow Rate of Mid-expiration (FEF 25-75 %), Peak Flow Rate (PEFR) were measured. When the values that were measured under 80% of expected were considered as significant deterioration in pulmonary functions. The results were shown as mean ± standard deviation. SPSS for Windows 10.0 programme was used for statistical analysis. We considered that $p<0,001$ is statistically significant.

Results

This study was performed on 26 of patients who underwent mitralvalve replacement in our clinic between January 2008 and June 2011 retrospectively. Patients' ages ranged from 24 to 74. 18 were female (69,2%) and 8 male (30,8%). Mean age was 55,88±14,52. Mitral valve area (cm^2) was 1,03±0,19 and median valve area was 1,03 cm^2 in preoperative period. Mean mitral gradient (mmHg) was 10,06±7,18 and median value was 8,0. Mean ejection fraction (EF %) was 54,38±11,52 and median value was 55,0. Mean pulmanory artery pressure (mmHg) was

57,12±18,98 and median value of pulmonary artery pressure was 60,0 in 9 of the patients with moderate tricuspid valve regurgitation and 17 of the patients with severe tricuspid valve regurgitation. Normal sinusal rhythms were observed in 3 patients and 23 patients had atrial fibrillation preoperatively. The data of pulmonary function tests were compared. Preoperative and postoperative measures of FVC, FEV1, FEV1/FVC, FEF 25-75 % and PEF were shown in Table-1 and Table-2 respectively.

Table – 1 Preoperative Spirometric Result

	PREOPERATIVE		
	Expected	Measured	%
FVC (L)	2,98 ± 0,87	2,17 ± 0,75	72,65 ± 18,67
FEV1 (L)	2,47 ± 0,72	1,64 ± 0,61	66,31 ± 15,96
FEV1/FVC (%)	78,12 ± 2,70	75,31 ± 10,21	
FEF25–75 % (L/sec)	3,17 ± 0,68	1,90 ± 0,75	60,85 ± 23,39
PEF (L/sec)	6,52 ± 1,37	3,83 ± 1,46	58,65 ± 18,60

Table – 2 Postoperative Spirometric Results

	POSTOPERATIVE	
	Measured	%
FVC (L)	2,58 ± 0,80	86,73 ± 10,99
FEV1 (L)	2,09 ± 0,68	84,73 ± 12,03
FEV1 / FVC (%)	81,58 ± 9,47	
FEF 25 – 75% (L/sn)	2,48 ± 0,88	78,42 ± 24,18
PEF (L/sn)	4,82 ± 1,39	74,04 ± 16,67

FVC, FEV1 FEV1/FVC, FEV1%, FEF 25-75% were within normal distribution. Whereas FVC% and PEF were not respected to normal distribution so that we performed Wilcoxon test. All the parameters were compared pre and postoperatively and were considered as significant due to p<0,001.

Discussion

The pulmonary process that occured in patients with mitral valve disease is called as "mitral lung". Small pulmonary trunk stenosis, restrictive and obstructive pulmonary function deteriorations are the most frequently seen conditions in patients with mitral lung (7-9).

Wood et al. reported 30% decrease in all lung volumes and flow rates in patients with mitral valve disease and twice increase in pulmonary resistance occured in the same group and this leads to 13% decrease in bronchial diameter (10).

Rhodes at al. showed in their study that the values FVC and FEV1 were decreased due to chronically
increased pulmonary venous pressures that lead to alveolar fibrosis (8).

It has been reported that bronchial hyper-reactivity occured in patients with mitral valve disease (11-14). On top of what has been reported, Cieslewicz at al. have indicated that this situation could be treated with steroid therapy (15).

In our study, we have identified a decrease in all expiratory volumes in 17 patients preoperatively. We suggested that the pulmonary dysfunction
which included both obstructive and restrictive pattern was due to the combination of decrease of both flow rates and volumes in these 17 patients. The other 9 patients developed obstructive and restrictive pattern or terminal pulmonary trunk obstruction separately. While preoperative FVC (L) and FEV1 (L) values were in normal levels, FEF 25-75 % (L/sec) values were under 80% of expected. We suggested that terminal pulmonary trunk obstruction has developed. These results indicated that terminal and/or common pulmonary trunk obstruction impairment as well as restrictive pulmonary trunk impairment occured in patients with mitral valve disease.

Morris et al. showed in their study that all lung volumes increased with the range of 30% in 4-6 weeks after mitral valve surgery in the patients with mitral valve disease (16).

Rhodes et al. reported that the improvement in values of postoperative FVC and FEV1 were statistically significant and there was no significant change in FEV1/FVC rates in

patients with mitral valve disease who undergone mitral valve surgery (8).

In our study, these parameters improved after 6 months of operation have remained below 30%, however has reached normal values. Statistically significant improvements were observed in FVC, FEV1, FEV1/FVC and FEF 25-75 % values. Range of FVC was 86,73 ± 10,99 %. Range of FEV1 was 84,73 ± 12,03 % and FEV1/FVC was 81,58 ± 9,47 % postoperatively.

Ohno et al. reported that there was significant improvement after valve surgery in patients with mitral valve disease in which the pulmonery hypertension and tricuspid valve regurgitation developed. However in patients with mitral valve disease in which the pulmonary hypertension and tricuspid valve regurgitation developed, there was no improvement in pulmonary functions (7).

In our study, although pulmonary hypertension and tricuspid valve regurgitation were observed in all patients preoperatively, statistically significant improvement in pulmonary functions were determined.

It has been reported that 5 of 17 patients inwhom all of the maximum expiratory volumes were decreased preoperatively, had the same findings in the postoperative period. Terminal pulmonary obstruction was continued in one patient and all parameters were improved in 9 patients. Isolated restrictive type of pulmonary dysfunction was present in 3 patients preoperatively and 2 of them were improved after mitral valve surgery. The study of Ohno et al (7). was not appropriate to analyse statistically because of number of individuals (5 patients). We think that our results were more reliable than Ohno's one because of the higher numbers of patients included to the study.

It has been suggested that the studies which were showing that there was no improvement in pulmonary functions after mitral valve surgery, were assessed incorrectly (8). The causes of wrong assessments are releated with incomplete knowledge of; presenting with cronical obstructive lung disease, smoking, performing the pulmonary function tests earlier than six months after surgery (the deteriorated pulmonary functions could not improved in early period after surgery because of the trauma of surgery). In addition, the absence of the assessment of effects of hemodinamics on pulmonary functions preoperatively and the absence of stratification of patients according to pulmonary disfunctions preoperatively (7,8).

Our study includes 6 months period of postoperative follow-up. Haemodynamic status of patients showed a common feature and the patients with cronical obtructive lung disease, history of smoking and history of previous pulmonary disease were excluded.

We concluded that the important part of the pulmonary dysfunctions due to mitral valve disease are reversible.

References

1.Roberts WC. Morphologic aspects of cardiac valve dysfunction. Am Heart J 1992; 123:1610-17.

2.Schwartz R, Myerson RM, Lawrence LT, et al. Mitral stenosis, massive pulmonary hemorrhage, and emergency valve replacement. N Engl J Med 1966; 275:755-67.

3.Carabello BA. Timing of surgery in mitral and aortic stenosis. Cardiol Clin 1991; 9:229-41.

4.Hygenholtz PG, Ryan TJ, Stein SW, et al. The spectrum of pure mitral stenosis. Am J Cardiol 1962; 10:773-96.

5.Alaa-Basiouni SM, Mohamed SB, Ali AH, et al. Effect of modified ultrafiltration on pulmonary function aftercardiopulmonary bypass. Chest 2005; 128:3447-53.

6.Kirklin JK, Blackstone EH, Kirklin JW. Cardiopulmonary bypass: Studies on its damaging effects. Blood Purif 1987; 5:168-87.

7.Ohno K, Nakahara K, Hirose H, et al. Effect of valvular surgery on overall and regional lung function in patients with mitral stenosis. Chest 1987; 92:224-28.

8.Rhodes KM, Evemy K, Nariman S. Effects of mitral valve surgery on static lung function and exercise performance. Thorax 1985; 40:107-12.

9.Techniques of evaluating pulmonary function in patients with cardiac disease in Hurst's The Heart, 8th ed., Schlant RC, Alexander RW, New York, McGraw-Hill, 1994; 441-45.

10.Wood T, McCleod P, Anthonisen NR. Mechanics of breathing in mitral stenosis. Am Rev Resp 1971; 104:52-60.
11.Rolla G, Bucca C, Caria E. Bronchial responsiveness in patients with mitral valve disease. Eur Respir J 1990; 3:127-31.
12.Snashall P, Chung KR. Airway obstruction and bronchial hyperresponsiveness in left ventricular failure and mitral stenosis. Am Rev Dis 1991; 144:945-55.
13.Nushimura Y, Maeda H, Yokoyama M. Bronchial Hyperreactivity in patients with Mitral Valve Disease. Chest 1990; 98:1085-90.
14.Hryniewiecki T, Rawczynska I, Malinowski R. Analysis of airway function in patients with mitral valve disease in various stages of progression. Przegl Lek 1999; 56:270-75.
15.Cieslewicz G, Juszezyk G, Foremmy J. Inhaled corticosteroid improves bronchial reactivity and decreases symptoms in patients with mitral stenosis. Chest 1998; 114:1070-74.
16.Valvular heart disease in Heart Disease: A textbook of cardiovascular medicine, 2nd ed., Braunwald E, Philadelphia, WB Saunders Company, 1995; 1007-26.