Per-operative Changes of Pulmonary Artery Pressure following Closed Mitral Commissurotomy

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Abstract

Key Words: Rheumaric fever, mitral valve, pulmonary artery pressure. **Background:** Mitral stenosis is often present with pulmonary hypertension. Closed Mitral Commissurotomy (CMC) is a treatment of choice for severe mitral stenosis. In this study, we examined the per-operative changes of pulmonary artery pressure following opening of stenosed mitral valve.

Methods: All these CMCs were performed routinely through the left antero-lateral thoracotomy (4^{th} intercostal space) and dilatation was done by metallic Tubb's Dilator. Peroperative left atrial and Pulminary Arterial pressures were measured before and after dilatation.

Results: 15 patients had undergone CMC. Following CMC, per-operative mean Pulmonary artery pressure was reduced from 45.5 ± 7.1 mm of Hg to 39.0 ± 8.8 mm of Hg (p=0.043). Mean left atrial pressure reduced from 35.9 ± 5.6 mm of Hg to 30.0 ± 9.1 mm of Hg (p=0.049). At three months follow up after closed mitral commissurotomy mitral valve area at echocardiography was found 2.29 ± 0.18 cm². There was no case of death after closed mitral commissurotomy. Mild mitral regurgitation occurred in 1 patient.

Conclusion: We conclude that there is immediate significant reduction of pulmonary Artery pressure following closed mitral commissurotomy. This reduction is apparently comparable with a similar reduction of left atrial pressure.

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Introduction:

Mitrial stenosis (MS) is highly prevalent in the developing countries because of its association with rheumatic fever, but it is also seen in developed countries.^{1,2} Excluding the developed economies, the global burden of rheumatic heart disease in the 5-14 year old children has been estimated to be 0.8 to 5.7/1000 with a median of 1.3/1000.^{3,4} A significant number of people in the developing countries do not receive adequate nutrition, sanitation, medical care and live in congested areas. Consequently, they suffer from recurrent attacks of rheumatic fever and severe mitral stenosis develops within 5 years of the initial episode.⁵ A substantial number of patients with mitral stenosis present at a late stage of the disease when the pulmonary artery pressure is very high. In the initial stages, pulmonary

hypertension may be confined to the pulmonary veins but later pulmonary arterial hypertension (PAH) supervenes. As the mitral valve orifice becomes narrower in mitral stenosis, pressure in the left atrium, pulmonary veins and pulmonary capillaries increases to maintain the blood flow at a normal level. The pressure rise in the pulmonary capillaries and veins gives rise to the symptoms of dyspnea, orthopnea, paroxysmal nocturnal dyspnea, pulmonary edema and hemoptysis. Later the pulmonary arterioles and small arteries in the lung become narrow due to histological changes in these vessels. Consequently, pulmonary artery pressure and pulmonary vascular resistance increase significantly. At this point, predominant symptoms are fatigue, exhaustion, weakness, tiredness with the appearance of cardiomegaly

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and hepatomegaly. The development of pulmonary arterial hypertension (PAH) has long been considered a risk factor for poor outcome in patients undergoing surgery.^{6,7}

Closed mitral commissurotomy (CMC) which was first reported by Cutler and Levine in 1923,8 had long been the treatment of choice for severe mitral stenosis. In South Asia the first ever digital closed mitral commissurotomy was performed by the famous Bengali surgeon Dr P K Sen at the King Edward Memorial Medical College, Bombay India in 1952.8 Some bold general surgeons performed at few digital mitral commissurotomy in late 1960s at Dhaka Medical College Hospital and the then Chest Hospital, which is the National Institute of the Diseases of Chest and Hospital today.9 After establishment of National Institute of Cardiovascular Diseases (NICVD) in 1978, CMC became the most frequently performed cardiac surgical procedure and remained so till the beginning of the Twenty-first century. With the advent of percutaneous transluminal mitral Commissurotomy (PTMC) this procedure has become near obsolete. However this time tested procedure is still being practiced in some centers. Despite being a city of five million inhabitants, Chittagong is yet to have any PTMC facilities and that's why CMC is still being performed in our center. This prospective study was conducted to assess the per-operative changes of pulmonary artery pressure following opening of stenosed mitral valve (following CMC).

Methods:

The study was carried out in the department of Cardiac Surgery, Chittagong Medical College & Hospital, Bangladesh from July 2013 to September 2016. This Prospective cohort study was conducted among the patients who had undergone CMC. Purposive sampling was done and 15 patients suffering from severe mitral stenosis with pulmonary hypertension were enrolled in the study. Patients with severe calcified mitral stenosis and/or with moderate to severe sub-valvular changes were excluded from this study. All these CMCs were performed routinely through the left antero-lateral thoracotomy (4th intercostal space) and dilatation of Mitral valve was done by Tubb's Dilator.

Peroperative pulmonary artery (PA) and left atrial (LA) pressures were measured before and after dilatation. Pressure was measured by inserting a needle within PA and LA. The needle was connected by an extension tube to a transducer. After proper de-aeration of the extension tube, pressures were measured which were displayed in the cardiac monitor. 3 months after CMC, echocardiographic examination with color Doppler was done to assess the cardiac status.

Results:

On the basis preoperative echocardiography, 15 patients were selected in the study who had severe mitral stenosis with pulmonary hypertension. These patients were between 16 and 45 years of age, with a mean age of 31.6 years. 3 patients were under 20 years of age, 5 patients were 21-30 years of age, 4 patients were 31-40 years of age and 3 patients were 41-50 years of age. Among the 15 patients 10 were female and 5 were male. The duration of symptoms ranged from 5 months to 4 years. The baseline characteristics of these patients are shown in Table I.

The mitral valve was approached through the left anterolateral thoracotomy (4^{th} intercostal space) and dilatation of Mitral valve was done by Tubb's Dilator. The mean operation time was 77 ± 20 minutes.

All of these patients had severe Mitral stenosis with pulmonary hypertension. We measured the pulmonary artery systolic, diastolic and mean pressure 10 minutes before and after dilatation. Before dilatation 7 patients had PA pressure >60/ 40 mm of Hg, 6 patients had PA pressure 50-60/ 30-40 mm of Hg and 2 patients had PA pressure <50/30 mm of Hg. The mean \pm SD value of mean PA pressure was 45.5 ± 7.1 mm of Hg before dilatation of Mitral valve. 10 minutes after dilatation we again measured the PA pressure. We observed that the PA pressures of all patients were reduced after dilatation. The mean \pm SD value of mean PA pressure was 39.0±8.8 mm of Hg. The reduction of mean PA pressure after dilatation was statistically significant (p=0.043).

We also measured the left atrial pressure simultaneously with PA pressure preoperatively.

Table-IBaseline characteristics of the study population (N=15).

Mean Age	31.6 years		
Sex	Male Female	5 patients 10 patients	
Mean duration of symptom	3.2 years		
NYHA class	Class II Class III	12 patients 3 patients	
Finding of Echocardiogram with colour Doppler Atrial fibrillation	Mean Mitral valve area	$0.78\pm0.14\mathrm{cm}^2$	
	Mean left atrial diameter	45.7 mm	
	Thrombus in left atrial appendage	Absent in all patients	
	Mean left ventricular diameter in diastole	35.3 mm	
	Condition of Mitral valve		
	Calcification	Calcification were present in 2 patients in the tip of anterior mitral leaflet	
	Subvalvular changes	Mild subvalvular changes were present in 2 patients Present in 6 patients	

The mean \pm SD value of mean LA pressure was 35.9 ± 5.6 mm of Hg before dilatation of Mitral valve. 10 minutes after dilatation the mean \pm SD value of mean LA pressure was 30.0 ± 9.1 mm of Hg. The reduction of mean LA pressure after dilatation was statistically significant (p=0.049).

Ionotropic support in the form of epinephrine, dopamine were required 3 patients post-operatively. There was no case of death in this series after closed mitral commissurotomy. Mild mitral regurgitation occurred in 1 patient following CMC. No patients had undergone re-exploration for bleeding after operation and there was no history of thromboembolic episodes post-operatively. The duration of ICU stay was

46±7 hours and hospital stay was 7.2±0.46 days.

At follow-up, patients were assessed clinically and by echocardiography. At one month follow-up, 6 patients (40%) were in NYHA class I and 9 (60%) patients were in NYHA class II. At three month follow-up, 13 patients (86%) were in NYHA class I and 2 (14%) patients were in NYHA class II. We measured the mitral valve area (MVA) before operation and 3 months after CMC by echocardiogram by a single cardiologist. The preoperative mean±SD value of MVA was 0.78±0.14 cm² and 3 months after CMC it was 2.29±0.18 cm². The difference between the two value was statistically highly significant (p=0.0001).

Table-II
Comparison of mean pulmonary artery pressure before and after CMC.

Mean PA pressure (mm Hg)	Before CMC	After CMC	p value	
Mean ± SD	45.5 ± 7.1	39.0±8.8	0.043	

Table-III
Comparison of mean left atrial pressure before and after CMC.

Mean LA Pressure (mm Hg)	Before CMC	After CMC	p value	
$Mean \pm SD$	35.9 ± 5.6	30.0±9.1	0.049	

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Table-IV
Comparison of mitral valve area before and after CMC.

MVA (cm2)	Before CMC	After CMC	p value
Mean ± SD	0.78 ± 0.14	2.29±0.18	0.0001

Discussion:

Pulmonary artery hypertension in mitral stenosis is more frequently seen in the developing country where the etiology is commonly rheumatic and presented at a younger age.⁵ The rise in left atrial pressure in mitral stenosis has been described as the most significant factor in the development of pulmonary hypertension with consequent vasoconstriction of the pulmonary arterioles.^{10,11} Some cases are further complicated by organic changes in the pulmonary vasculature, but these changes are moderate and are probably reversible after mitral valve surgery.^{12,13}

Before widespread availability of PTMC, closed mitral commissurotomy was the treatment of choice in patients presenting with mitral stenosis with no or mild mitral insufficiency and a flexible mitral valve, free of calcification. 14 It usually allows a better decompression of the left atrium and pulmonary vasculature. 15 In our study we observed that the mean \pm SD value of mean PA pressure was 45.5 ± 7.1 mm of Hg before dilatation of mitral valve. 10 minutes after dilatation the PA pressure of all patients was dramatically reduced and the mean \pm SD value of mean PA pressure was 39.0±8.8 mm of Hg. The reduction of mean PA pressure after dilatation was statistically significant (p=0.043). At the same time the mean \pm SD value of mean LA pressure was reduced after mitral commissurotomy from 35.9 ± 5.6 mm of Hg to 30.0±9.1 mm of Hg and the reduction of mean LA pressure after dilatation was also statistically significant (p=0.049). This sudden reduction in PA pressures may occur due to acute reduction in LA pressures and reversal of pulmonary vasoconstriction. The increase in cardiac index is attributed to the improvement in right ventricular function.

In some studies which were conducted between 1965-1975 showed that patients with pulmonary hypertension had two to three times greater risk in the early postoperative period than those with lesser degrees of pulmonary hypertension undergoing identical surgical procedures. ^{16,17} Furthermore, the long-term prognosis of these patients has been described as poor. ¹⁸ However, in recent studies it was observed that the postoperative morbidity and mortality after valvular surgery became low and survival after mitral valve surgery is slightly influenced by the severity of pulmonary hypertension. This support the view that a commissurotomy should be performed whenever indicated and we found that all the patients are in NYHA class I or II after surgery.

Most studies showed no difference in survival rates between the closed and open mitral commissurotomy. ¹⁹⁻²¹ Scalia and colleagues showed identical survival rates using both techniques. ²¹ Hickey and associates were also able to demonstrate that the operative technique was not a risk factor for long-term survival. ¹⁹ Our study also showed that closed mitral commissurotomy is an effective procedure to treat the mitral stenosis without any mortality and morbidity.

Many researchers believe that mitral commissurotomy should be performed as early as possible to prevent progressive valve changes, which make reconstruction impossible. ²²⁻²⁴ Such early surgical intervention could possibly prevent serious secondary changes such as left atrial hypertrophy, atrial fibrillation, pulmonary hypertension, tricuspid insufficiency and the risk of thromboembolism. 24 Thus, early operation and the proper selection of patients should be goal when performing a major commissurotomy. In well-selected patients with pure mitral stenosis and no leaflet calcification, closed mitral commissurotomy still remains a valid surgical option.

Conclusion:

We conclude that in well-selected patients with pure mitral stenosis and no leaflet calcification, closed mitral commissurotomy still remains a valid surgical option and there is immediate significant reduction of pulmonary artery pressure following closed mitral commissurotomy. This reduction is apparently comparable with a similar reduction of left atrial pressure.

Conflict of Interest - None.

References:

- Padmavati S. Rheumatic fever and rheumatic heart disease in India at the turn of the century. *Indian Heart Journal* 2001; 53: 35–37.
- Carroll JD, Feldman T. Percutaneous mitral balloon valvotomy and the new demographics of mitral stenosis. JAMA 1993; 270: 1731–1736.
- Carapetis JR, Steer AC, Mulholland EK, Weber M. The global burden of group A streptococcal disease. *Lancet Infect Dis* 2005; 5: 685-694.
- Carapetis JR. Rheumatic heart disease in Asia. Circulation 2008: 118: 2748-2753.
- Padmavati S. Present Status of Rheumatic Fever and Rheumatic Heart Disease in India. *Indian Heart Journal* 1995; 47: 395-398.
- Chaffin JS, Daggett WM. Mitral Valve Replace-ment: A Nineyear Follow-up of Risks and Survivals. Ann Thorac Surg 1979; 27: 312-319.
- Ward C and Hancock BW. Extreme Pulmonary Hypertension Caused by Mitral Valve disease- Natural History and Results of Surgery. Brit Heart J 1975: 37: 74-78
- Naidu KV. Presidential address. 50 years of cardiac surgery in India. Indian J Thorac Cardiovasc Surg 1996; 12(1):1-6.
- Hosain N. The early days of Cardiac Surgery in South Asia: The history and heritage. Ann Thorac Surg 2017;104:361-366.
- Cutler EC, Lewine SA. Cardiotomy and valvulotomy for mitral stenosis- Experimental observations and clinical notes concerning operated cases with recovery. Boston Med Surg J 1923; 188: 1093-1097.

- 11. Wood P. Pulmonary hypertension with special reference to the vaso constrictive factor. Brit Heart J 1958: 20: 557-560
- Harris P, Heath D. The Human Pulmonary Circulation. Williams and Wilkins, Baltimore: Livingstone, Edinburgh.1977: 225.
- Ramirez A, Grimes ET, Abelmann HWH. Regression of pulmonary vascular changes following mitral valvuloplasty. Am J Med 1968; 45: 975-979.
- Eguaras MG, Jimenez MA, Calleja F. Early open mitral commissurotomy: long-term results. J Thorac Cardiovasc Surg 1993; 106: 421–426.
- Braunwald E, Braunwald NS, Ross J, Morrow AG. Effects of mitral-valve replacement on the pulmonary vascular dynamics of patients with pulmonary hypertension. N Engl J Med 1965; 273: 509-514.
- Starr A, Herr RH, Wood JA. Mitral valve replacement. J Thorac Cardiovasc Surg 1967; 54: 333-337.
- Najafi H, Dye WS, Javid H, Hunter JA, Ostermiller WE, Julian 0C. Mitral valve replacement. Am J Cardiol 1969; 24: 386-390.
- Barclay RS, Reid JM, Stevenson JG, Welsh TM, McSwan N. Long-term followup of mitral valve replacement with Starr-Edwards prosthesis. *Brit Heart J* 1972; 34: 129-134.
- Hickey MSJ, Blackstone EH, Kirklin JW, Dean LS. Outcome probabilities and life history after surgical mitral commissurotomy: implications for balloon commissurotomy. J Am Coll Cardiol 1991; 17: 29–42.
- Molajo AO, Bennett DH, Bray CL. Actuarial analysis of late results after closed mitral valvotomy. *Ann Thorac Surg* 1988; 45: 364–369.
- Scalia D, Rizzoli G, Campanile F. Long-term results of mitral commissurotomy. J Thorac Cardiovasc Surg 1993; 105: 633–642.
- Spencer FC. A plea for early, open mitral commissurotomy. *Am Heart J* 1978; 95: 668–670.
- Eguaras MG, Luque I, Montero A. Conservative operation for mitral stenosis. J Thorac Cardiovasc Surg 1988; 95: 1031–1037.
- Laschinger JC, Cunningham JN, Baumann FG. Early open radical commissurotomy: surgical treatment of choice for mitral stenosis. Ann Thorac Surg 1982; 34: 287–296.