

Mitral Regurgitation Associated with Secundum Atrial Septal Defect

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Abstract

Introduction: Mitral regurgitation associated with secundum atrial septal defect is not uncommon. We reported our experiences and also reviewed its etiologies and managements in the literatures.

Patients and Methods: Mitral regurgitation was found in 13% (12 patients) of all patients with secundum atrial septal defect (93 patients) who were operated on during 9-year period at Yala Hospital. The etiologies were as follows : rheumatic valve 3, prolapsed anterior leaflet 2, congenital abnormality 2, specific pathology complex 4, and chronic infective endocarditis 1. All patients underwent closure of secundum atrial septal defect with mitral valve repair.

Results: There was one death and one serious post-operative complication. On follow-up, the echocardiograms showed no mitral regurgitation in 10 patients and mild mitral regurgitation in 1 patient.

Conclusion: Mitral regurgitation associated with secundum atrial septal defect could exist as an co-existent lesion or as the result of hemodynamic change occurred in secundum atrial septal defect. Its recognition is important and most of them could be repaired with satisfactory results.

Key words: Mitral regurgitation, secundum atrial septal defect

INTRODUCTION

The association of a secundum atrial septal defect and mitral regurgitation is not uncommon. Reports have attributed mitral regurgitation in these patients to disease of mitral valve prolapse,¹⁻⁴ congenital valve deformity,⁵ rheumatic valve,⁶ and specific pathophysiology complex.⁷ The importance of its recognition and management was also emphasized.^{6,8,9} We reported here our experiences of this combined lesions regarding its incidence, etiologies, and results of management.

PATIENTS AND METHODS

From January 2001 to July 2010, 12 patients with secundum atrial septal defect associated with mitral regurgitation were operated. This represented 13% of all patients with secundum atrial septal defect (93 patients) who were operated during the same period. Patients with secundum atrial septal defect combined with other congenital cardiac defects were excluded. The diagnosis and severity of mitral regurgitation was made by pre-operative transthoracic echocardiogram. The etiologies of mitral regurgitation was made by

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intra-operative examination.

RESULTS

1. Incidence

The details of clinical characteristics and operations were shown in Table 1. There were 3 males and 9 females, age ranged from 11 to 59 years. The severity of mitral regurgitation by transthoracic echocardiogram was moderate in 11 patients and mild in 1 patient.

2. Etiology

The mitral valves were carefully assessed intra-operatively. The mechanism of mitral regurgitation was evaluated and the mitral valve apparatus was inspected and examined for any abnormality that could be the cause of mitral regurgitation. The

etiologies of mitral regurgitation in these 12 patients could be as follow : rheumatic heart disease in 3 patients, prolapsed anterior leaflet in 2 patients, congenital abnormality in 2 patients, specific pathophysiology complex (7) in 4 patients, and chronic bacterial endocarditis in 1 patient. Details of the mitral valve abnormalities were shown in Table 1. For rheumatic heart in origin, the posterior mitral leaflet and its chordae were thickened and retracted as shown in Figure 1 and Figure 2. For prolapsed anterior leaflet, the posterior mitral leaflet looked normal but the anterior mitral leaflet were slightly prolapsed. For congenital abnormality, one had cleft at posterior mitral leaflet and the other had small or underdeveloped posterior mitral leaflet. For chronic bacterial endocarditis, there was ruptured chordae of anterior leaflet at the medial side. For the specific pathophysiology complex, there was a thickening at

Table 1 Clinical characteristics, mechanism and etiology of MR, operative methods and results

No	Sex	Age year	MR Grading	Abnormality of Mitral Valve	Etiology MR	Repair Method	TVA done	Post-op Result	Follow up Result
1	F	15	Moderate	Prolapsed AML	Prolapsed AML	Commissuroplasty, Ring	no	no MR	no MR
2	F	14	Moderate	Prolapsed AML	Prolapsed AML	Mobilised PML, Ring	Yes	no MR	no MR
3	M	16	Moderate	Restricted P2	ASD, MR Complex	Ring	no	no MR	no MR
4	F	22	Moderate	Retracted P1	Rheumatic	Mobilised P1, Papillotomy, Ring	Yes	no MR	no MR
5	F	46	Moderate	Restricted PML	Rheumatic	Mobilised PML, Ring	Yes	Dead	
6	M	41	Moderate	Ruptured Chordae A3	Chronic infective endocarditis	Artificial Chordae A3, Ring	Yes	no MR	no MR
7	F	49	Moderate	Restricted P2	ASD, MR Complex	Papillotomy, Ring	Yes	no MR	no MR
8	F	11	Moderate	Cleft PML	Congenital	Suture cleft, Ring	Yes	no MR	mild MR
9	F	25	Moderate	Small PML	Congenital	Mobilised PML, Plication P1, no Ring	Yes	Cerebral emboli, no MR	no MR
10	M	14	Moderate	Restricted P2	ASD, MR Complex	Ring	Yes	no MR	no MR
11	F	59	Mild	Restricted P2	ASD, MR Complex	Plication P2	Yes	no MR	no MR
12	F	23	Moderate	Restricted PML	Rheumatic	Mobilised PML, Ring	Yes	no MR	no MR

F = Female, M = Male , AML = Anterior mitral leaflet, PML = Posterior mitral leaflet

TVA = Tricuspid valve annuloplasty, MR = Mitral regurgitation

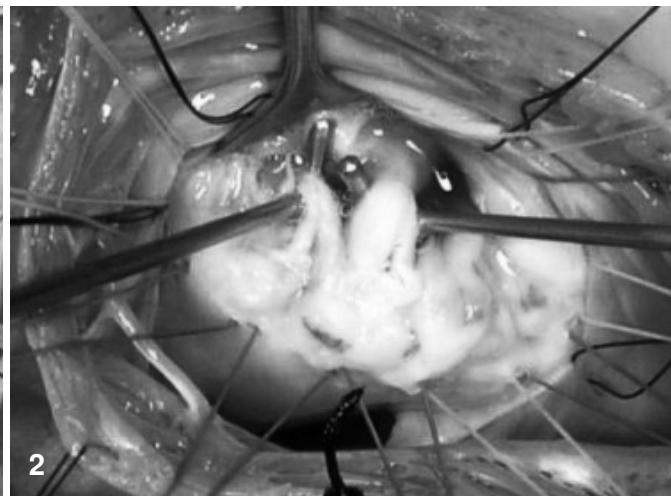


Figure 1 & 2 Demonstrated mitral regurgitation from rheumatic valvular disease associated with secundum atrial septal defect. The posterior mitral leaflet and its chordae were thickened and retracted.

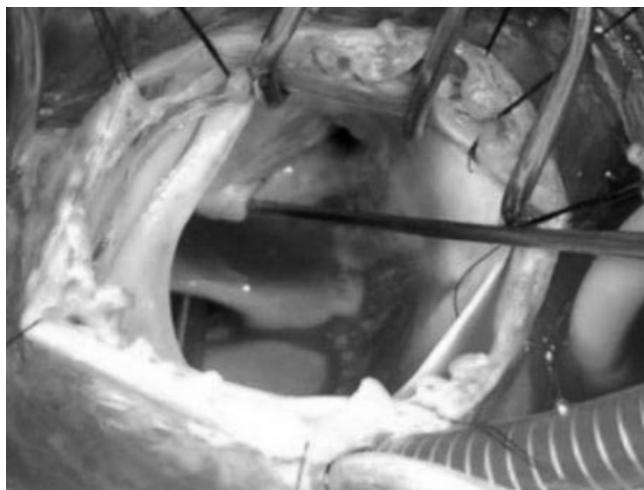


Figure 3 Demonstrated mitral regurgitation from specific pathology as described by Furuta and associated with secundum atrial septal defect. There was thickening at the posterior-medial half of anterior and posterior mitral leaflet as described by Furuta.⁷

the posterior-medial half of anterior and posterior mitral leaflet as described by Furuta,⁷ as shown in Figure 3.

3. Operations and Results

All patients underwent closure of secundum atrial septal defect with mitral valve repair. Details of the repair procedures and results were shown in Table 1. All patients except one had flexible rings implantation. There were one death and one serious post-operative complication. One early death from low cardiac output

was a 46-year-old woman with functional class 4 preoperatively. One case with postoperative cerebral emboli occurred in a 25-year-old woman. On follow-up, ranged from 2 to 60 months (mean 33 months), the echocardiograms showed no mitral regurgitation in 10 patients, and mild mitral regurgitation in one patient.

DISCUSSION

Mitral regurgitation associated with secundum atrial septal defect was not uncommon. Its incidence ranged from 4% to 9.1% (Table 2). Its recognition is important because the clinical course of mitral regurgitation is altered by the presence of an atrial septal defect. Some patients with severe mitral regurgitation may not manifest symptoms of mitral regurgitation because the atrial septal defect unloads the left atrium. If residual mitral regurgitation is significant following atrial septal defect closure, increased left atrial and pulmonary venous pressure develop and may produce or increase symptom. Closure of the atrial septal defect without repair of mitral regurgitation so might increase postoperative morbidity and mortality rates.

The causes of mitral regurgitation associated with secundum atrial septal defect can be classified into extrinsic and intrinsic factors. The extrinsic factors maybe the coexistent disease such as rheumatic valve disease or infective endocarditis which are also common disease in the developing country. For the intrinsic

Table 2 Reported cases of mitral regurgitation associated with secundum atrial septal defect in the literatures

Year published	Author	Total cases (incidence%)*	Etiologies	Treatment	Result
1974	Hynes (10)	46 (6 %)	Rheumatic 6 Prolapsing leaflet 8 Ruptured chordae 3 Not definite 29	None 26 Repair 16 Replacement 2	Early death 1 Late death 3
1975	Murray (11)	4	Rheumatic 1 Congenital cleft 1 SBE 1 Traumatic 1	Repair 4	No death
1977	Davies (5)	5	Cleft	Repair 5	No death
1977	Pansingha (8)	12 (5.6%)	Rheumatic 3 Ruptured chordae 1 Not definite 8	Repair 4 Replacement 8	Early death 1 Late death 1 (Both from replacement)
1978	Shigenobu (9)	13	Not definite 13	Repair 12 Replacement 1	Early death 2 (repair) Late death 1 (replacement)
1979	Boucher (6)	10 (4%)	Rheumatic 3 Not definite 7	Replacement 4 (Redo 1)	Early death 1 Late death 1
1982	Furuta (7)	27 (9.1%)	ASD - MR complex 27	Repair 21 Replacement 3 None 3	Early death 1 (repair) Late death 2 (replacement)

*from all patients with isolated secundum atrial septal defect operated during the same period

SBE = Subacute bacterial endocarditis

factors, there were many evidences from the literatures that secundum atrial septal defect itself could give rise to mitral regurgitation. The incidence of mitral valve prolapse in patients with atrial septal defect has been reported for a several decades.¹ By angio-graphic and echocardiographic studies, Ballester et al² proposed that prolapse of the mitral valve in patients with secundum atrial septal defect may be related to the distorted left ventricular shape and small left ventricular volume. Davies,¹² by pathological studies, demonstrated thickening of the medial half of the anterior cusp and some fusion of adjacent chordae. He also demonstrated histologically that the lesion was a surface fibrosis without vascularisation or myxomatous change to suggest a rheumatic or myxomatous origins. He concluded that it was secondary valve changes caused by abnormal cusp movement and resultant valve trauma presumably related to increased flow and altered left ventricular geometry found in secundum atrial septal defect.

There was one interesting hypothesis which was studied and proposed by Furuta and colleagues in 1982.⁷ By intra-operative studies, they found abnormal fibrous thickening at the medial half of mitral valve at

which leakage of the valve occurred. They proposed the specific pathophysiology called "ASD, MR complex" that friction between anterior and posterior mitral leaflets due to abnormal left ventricular motion secondary to the right ventricular volume overload might be responsible to the genesis of this lesion. So the pathogenesis of mitral regurgitation in ASD could be due to abnormal hemodynamic change in ASD itself, just similar to the aortic regurgitation caused by VSD.

In our experience the incidence of mitral regurgitation associated with ASD was 13% and the causes were varied. Such "ASD, MR complex" was presented in about 30% in our patients. Rheumatic valvular heart diseases were still common problem in our area, so it could have the opportunity to co-exist with ASD. Prolapse of mitral valve was also found in our patients. However, these were based on echocardiographic and intra-operative findings which might not be true. So, the exact causes of mitral regurgitation in these patients might be different from our diagnosis.

From whatever cause it might be, most of the mitral valves in mitral regurgitation associated with

secundum atrial septal defect could be repaired with satisfactory results, as reported from many literatures (details demonstrated in Table 2). This might be due to usually not severe mitral valve pathology in these combined lesions. Many authors^{7,9,10} strongly urge mitral valve repair for patients with mitral regurgitation at the time the secundum atrial defect is repaired.

In conclusion, mitral regurgitation associated with secundum atrial septal defect could exist as an co-existent lesion or as the result of hemodynamic change occurred in secundum atrial septal defect itself. Its recognition is important and most of them could be repaired with satisfactory results.

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