Arch Jpn Chir 54(3), 222～229, Mai, 1985

臨　　床

Surgical Treatment of Mitral Regurgitation Casued by Chordal Rupture

Yoshifumi Okamoto, Atsushi Awazu, Masataka, Osaragi, Toyo Nihei*, Shigefumi Morioka*, Katsutoshi Moriyama*, Yutaka Watanabe**

Shimane Medical University 1st Department of Surgery
*4th Department of Internal Medicine
**Ohtsu Red Cross Hospital
Received for Publication, Feb. 28, 1985.

Abstract

We experienced 18 cases of mitral regurgitation due to ruptured chordae tendineae and reviewed these cases with respect to clinical picture and surgical procedure.

Etiologic factors for chordal rupture were bacterial endocarditis in 3 cases, Marfan's syndrome in 1 case, corrected transposition of the great arteries in 1 case, degenerative in 10 cases and unknown in 3 cases. The site of chordal rupture was near the left anterior leaflet in 8 cases, near the right anterior leaflet in 5 cases, and the posterior leaflet in 5 cases.

Mitral valve repair was performed in 8 cases, in which the reoperation was necessary in 3, because of residual or recurrent incompetence within 2 years. Valve replacement was performed in 10 cases, reoperated in one caused by a dehiscence of prosthesis.

When the affected valvular tissue is thin and fragile, little can be expected from valvuloplasty and, accordingly, valve replacement is recommendable. However, in attempting valve replacement contrivances are needed to prevent dehiscence due to disruption of the remaining valvular tissue.

We performed valve replacement with a low profile bileaflet St. Jude Medical prosthesis without removal of the valve and subvalvular tissue in 8 cases of degenerative rupture of chordae tendineae of the mitral valve (of unknown etiology) with satisfactory results.

Introduction

There are various diseases that can be a precipitating cause of chordal rupture, including bacterial endocarditis, rheumatic fever, thoracic trauma, myocrdial infarction, Marfan's syndrome and mitral commissurotomy. However, many cases of degenerative (or spontaneous) chordal

Key words: Ruptured chordae tendineae, Valve repair, Valve replacement, Degeneration, Reoperation.
索引語：腱索断裂，弁形成術，人工弁置換術，組織変性，再手術．
Present address: Department of Cardiovascular Surgery, Faculty of Medicine, Kyoto University, Kyoto, Japan.

rupture have been also reported.

When cardiopulmonary bypass became reasonably safe and prosthetic devices that guaranteed immediate competence were developed, valve replacement became the standard treatment for mitral disease. Recently, the progress in myocardial protection that offers a dry, quiet operative field and allows for long unhurried procedures has opened the possibility of new reconstructive methods tailored to the specific lesion in each case. We considered to review our experience of surgical treatment of chordal rupture.

Patients

In the last 9 years, from April 1975, we experienced 18 cases of mitral regurgitation due to ruptured chordae tendineae. Clinical features of the 18 patinets were as indicated in Table 1. This series comprised 11 male and 7 female with an age range of 25 to 69 years (mean 49).

The length of time elapsing from the clinical manifestation until operation ranged from 4 months to 10 years with an average of 3.6 years. In cases 2 and 3 the patients were noticed of their having cardiac murmurs at the age of 10 and 15 years, respectively, and diagnosed as having a mitral valvular disease. It was difficult, therefore, to determine whether the cardiac murmurs were caused by rupture of the chordae tendineae. Case 4 was corrected transposition of the great arteries; the patient was noticed of his having cardiac murmurs 13 years previously.

Judging from the history of present illness, 8 of the entire 18 patients were thought to have had a sudden onset of disease characteristic of chordal rupture with manifestation or accentuation of an apical systolic murmur. In other cases it was impossible to determine the time of occur-

Table 1.

Patient No.	Age (yr) and Sex	History	NYHA class	Cardiothoracic index (%)	Pulmonary hypertension
1	62/M	10 yr	III	56	−
2	28/M	6 mo	II	53	+
3	28/F	8 yr	II	60	−
4	50/M	10 yr	III	45	+
5	53/M	4 mo	III	55	−
6	49/M	4 yr	III	60	++
7	25/M	4 mo	IV	60	++
8	50/F	3 yr	III	60	−
9	56/F	2 yr	III	50	−
10	41/M	6 mo	IV	53	++
11	51/M	4 yr	IV	65	++
12	49/F	2 yr	III	63	+
13	69/F	9 yr	III	50	−
14	38/M	2 yr	III	55	−
15	67/F	3 yr	III	63	+
16	52/F	1 yr	II	55	−
17	61/M	2 yr	III	55	+
18	57/F	2 yr	II	50	−

rence of chordal rupture.

Electrocardiography revealed atrial fibrillation in 6 patients while normal sinus rhythm in others. Cardiac catheterization disclosed marked pulmonary hypertension in cases 6, 7, 10 and 11, moderate pulmonary hypertension in cases 2, 4, 12, 15 and 17 and almost normal pulmonary arterial pressure values in the remaining cases. Left ventriculography revealed mitral regurgitaion of grade III–IV of Sellers' classification.

As complications there were corrected transposition of the great arteries and complete atrioventricular block in case 4, aortic regurgitation in case 7, tricuspid regurgitation in cases 7, 8 and 11 and myocardial infarction in case 16.

Operative Findings and Procedures

The site of chordal rupture was near the left anterior cusp in 8 cases, near the right anterior cusp in 3 cases, near the medial scallop in 3 cases, near the right middle scallop in 2 cases and at the anterior cusp (which side undefined) in 2 cases.

The cause of chordal rupture was bacterial endocarditis in 3 cases, Marfan's syndrome in 1 case and corrected transposition of the great arteries in 1 cases. In 10 cases the chordal rupture was categorized as degenerative, unknown in 3 cases.

Case 4 was of rupture of the left atrioventricular valve due to corrected transposition of the great arteries. In case 7 there was a marked deterioration of the mitral valve with a perforation of the anterior leaflet in the presence of bacterial endocarditis. Cases 8 and 16 had thickened mitral valve leaflets with dilatation of the annulus and organization to torn ends of the chordae tendineae; these pathologic changes indicated further studies to elucidate their true etiology.

Table 2.

Patient No.	Operative Findings (Ruptured)	Etiology
1	left anterior cusp	degenerative
2	left anterior cusp	unknown
3	right anterior cusp	Marfan syndrome
4	anterior cusp	corrected TGA
5	anterior cusp	infective endocarditis
6	medial scallop	degenerative
7	left anterior cusp	infective endocarditis
8	medial scallop	unknown
9	medial scallop	degenerative
10	right middle scallop	degenerative
11	left anterior cusp	infective endocarditis
12	left anterior cusp	degenerative
13	left anterior cusp	degenerative
14	left anterior cusp	degenerative
15	right anterior cusp	degenerative
16	right anterior cusp	unknown
17	left anterior cusp	degenerative
18	right middle scallop	degenerative

Table 3. Late follow-up results

Patient No.	Follow-up (yr)	NYHA class	Systolic murmur (grade)	Echocardiography		Other finding
				Prolapse	MVA (cm^2)	
1	5	I	0		3.2	Reoperation
2	6	I	0		3.0	Reoperation
3	4	I	0		3.5	Reoperation
4	5	I	0		2.6	
5	8	I	0		3.8	
6	7	I	2			Reoperation
7	8	II	0		4.0	
8	4	I	1	No	2.8	
9	4	I	0	No	3.7	
10	3	I	2	No	3.2	
11	3	II	0		3.4	
12	2	I	0		2.7	
13	2	II	0			
14	1	II	0			
15	1	II	0			
16	1	II	0		3.0	
(17)						died of acute hepatitis
18	1	II	0	No		

When ruptured chordae tendineae were small in size or number, valve repair was done, with or without annuloplasty.

Reoperations, however, were necessary in 4 patients within 2 years. There were residual incompetence after valve repair in case 1 and 2, reccurent incompetence in case 3, After valve replacement in case 6 dehiscence of prosthesis occurred. In cases, in which the valvular tissue appeared fragile (with dilatation of the annulus and thinning and elongation of the chordae tendineae and leaflets) mitral valve replacement was attempted. Uses for this purpose were low profile, bi-leaflet St. Jude Medical Prostheses, which were transplanted without removal of the affected mitral valve and subvalvular apparatus. This operative procedure allows to make a firmer support for the prosthesis and at the same time permit preservation of papillary muscles which would favor functioning of the left ventricle. Valve replacement by this procedure was done in 8 cases, using #25 prosthesis in 1, #29 in 2 and #27 in 5 cases.

In case 7 aortic valve replacement was performed simultaneously for concurrent aortic regurgitation, while in cases 7, 8 and 11 annuloplication with Bex ring was concomitantly done for tricuspid regurgitation. In case 4 the patient had an artificial pacemaker implanted as mitral incompetence led to complete atrioventricular block. Atrial fibrillation persisted after operation in all cases but one (Case 16), There were no operative deaths, nor did any fatalities occur during a postoperative follow-up period but one of acute hepatitis (3 months to 8 years). Late follow up is summarized in Table 3.

Discussion

Mitral regurgitation (MR) caused by ruptured chordae tendineae is characterized clinically by a sudden onset of a cardiac murmur and heart failure, sinus rhythm on ECG and auscultatory findings of a fourth heart sound and a holosystolic murmur, sometimes decrescendo late in systole, similar to that of aortic stenosis. Acute MR is said to produce more severe pulmonary hypertension than the chronic variety. However, MR depends for its following an acute course upon the number of chordae tendineae torn, their location, the amount of regurgitation into the left atrium and left atrial compliance.

A review of the history of present illness revealed 9 of the entire 18 cases to have developed acute symptoms characteristic of chordal rupture; in another, cardiac murmur that suddenly occurred during treatment of bacterial endocarditis and echocardiographic findings proved to be diagnostic. In the rest of cases it was impossible to determine the time of occurrence of chordal rupture. Marked pulmonary hypertension was present in 4 cases, with one of them having concurrent perforation of the affected valve. It is said that rupture even of the same chordae tendineae may produce a different clinical picture. An extra volume load, if suddenly imposed on the left side of the heart, may conceivably be mitigated by a compensatory mechanism in cases with preexisting valvular or heart disease or compensated for to a more or less extent in young patients.

As regards the site of lesion, chordae tendineae of the anterior leaflet are torn more commonly in the presence of bacterial endocarditis or rhematic disorders, while degenerative (or spontaneous) tends to be associated with rupture of the posterior leaflet[10].

The description by McGoon[16] of plication of the posterior cusps to approximate valve tissue supported by intact chordae showed the possibility of avoiding valve replacement in patients with ruptured mitral valve chordae. Manhas and colleagues[15] modified this technique by performing triangular excisions of the part of the cusp served by the ruptured chordae. Quadrantal excision of posterior cusp have been added to the original technique[1,8,19]. The results of posterior chordal rupture repairs by these methods are good[6,13]. But in cases of ruptured anterior cusp chordae have been reported to be of limited value. Shore and colleagues[20] gave up attempting repairs of ruptured anterior cusp chordae with V excision and annuloplasty. Carpentier and associates[2] have classified the indication of ruptured chordae; rupture of the main chordae of the anterior leaflet is a contraindication, rupture of the paramedial anterior cusp chordae only a relative indication and rupture of a third of the posterior cusp chordae or fewer is a primary indication. The excision technique has limited value for anterior chordal rupture because the base of the anterior cusp has a fixed dimension and cannot easily be shortened and because the free edge of the anterior cusp is convex and variable in depth, so that removal of a portion of it and the displacement of the remaining cusp when the ensuing defect is stitched may result in failure to meet the concave posterior cusp despite annuloplasty[7]. Frater[7] has described in detail about reproducible replacement of ruptured chordae with tanned xenograft pericardium. Carpentier and co-workers[2,3] have made the most original and extensive

contribution to dealing with elongated chordae with their various shortening procedures. But we have found chordal replacing or shortening very difficult to do when confronted with the elongated, stretched and thinned chordae of degenerative disease. In such an instance valvuloplasty is infeasible too, or even if feasible, very much likely to be followed by a recurrence.

A valve-conserving operation, if proves to be satisfactory, would, of course, be the treatment of choice. As mentioned earlier, however, the cause of chordal rupture is quite diverse and, moreover, a predominant etiologic role was played by bacterial endocarditis and rheumatic disorders before the advent of antibiotics, while in these latest years degenerative rupture (or spontaneous) is on the increase. It is natural, therefore, that due consideration should be paid to the cause of chordal rupture in making a choice of operative procedure.

From the fact that many of patients with chordal rupture are in their forties of fifties in male it seems probably that some degenerative changes with age of the chordae tendineae and leaflets of the mitral valve play a significant etiologic role, The designation degenerative mitral valve disease encompasses a number of syndromes and probably some variations in histological appearance as well.

When the affected valvular tissue is thin and fragile, little can be expected from valvuloplasty and, accordingly, valve replacement is recommendable. However, in attempting valve replacement contrivances are needed to prevent dehiscence due to disruption of the remaining valvular tissue[4,5,11,12,17].

We performed valve replacement with a prosthesis without removal of the valve and subvalvular tissue in 8 cases of degenerative rupture or chordae tendineae of the mitral valve (including unknown etiology) with satisfactory results. In our procedure, surgical removal of valvular tissue is limited to ruptured chordae tendineae and prolapsing portion of the leaflet involved and a low profile, bi-leaflet St. Jude Medical prosthesis is sutured to the annulus by interrupted U sutures with pledget. By suturing the prosthesis to the remnant valve with its two leaflets apposed to the anterior and posterior cusps, respectively, it is possible to forestall impaired movability of the prosthetic valve which could otherwise become a problem.

Moreover, the procedure which preserves the papillary muscles will afford the advantage of sustaining left heart function. Previously RASTELLI[18], LILLEHEI[14] and others disputed about resection of papillary muscles and left heart function in mitral valve replacement.

To the best of our knowledge no reports in favor of valve replacement have been made of late presumably and partly because of gratifying results of valve repair reported by JACOB[9] Nevertheless, emphasis still has to be placed on the fact that cases do exist in which valve repair is impracticable for morphological reasons or where degenerative transformation and fragility of the valve involved make this modality of surgical treatment quite difficult to perform. Further in-depth studies of long-term results of operation for mitral regurgitation in relation to the etiology of chordal rupture seem to be indicated.

References

1) Carpentier A, Relland J, et al: Conservative management of the prolapsed mitral valve. Ann Thorac Surg **26**: 294–302, 1978.
2) Carpentier A, Chauvaud S, et al: Reconstructive surgery of mitral valve incompetence. J Thorac Cardiovasc Surg **79**: 338–348, 1980.
3) Carpentier A, Deloche A, et al: A new reconstructive operation for correction of mitral and tricuspid insufficiency. J Thorac Cardiovasc Surg **61**: 1–13, 1971.
4) Caves PK, Paneth M: Replacement of the mitral valve, aortic valve and ascending aorta with coronary transplantation in a child with the Marfan's syndrome. Thorax **27**: 58–65, 1972.
5) Cooley DA: Mitral insufficiency due to myxomatous transformation—floppy valve syndrome—. J Cardiovasc Surg **13**: 346–350, 1972.
6) Duran CG, Pomar JL, et al: Conservative operation for mitral insufficiency. J Thorac Cardiovasc Surg **79**: 326–337, 1980.
7) Frater RWM, Gabbay FRS, et al: Reproducible replacement of elongated or ruptured mitral valve chordae. Ann Thorac Surg **35**: 14–28, 1983.
8) Jacoub M, Halim M, et al: Surgical treatment of mitral regurgitation caused by floppy valves: Repair versus replacement. Circulation **64**: 210, 1981.
9) Kremkau EL, Gillerston PR, et al: Acquired nonrhematic mitral regurgitation. Prog Cardiovasc Dis **15**: 403–410, 1972.
10) Kaku R: Mitral insufficiency in an incomplete form of Marfan's syndrome corrected with valve replacement. J Cardiovasc Surg **16**: 95–99, 1975.
11) Kiser JC, Martin FE, et al: Mitral insufficiency in Marfan's syndrome—surgical correction—. JAMA **213**: 1039–1040, 1970.
12) Lessana A, Herreman F, et al: Hemodynamic and cineangiographic study before and after mitral valvuloplasty. Circulation 64: Supple. **2**: 195–202, 1981.
13) Lillehei, CW Levy MI, et al: Mitral valve replacement with preservation of papillary muscles and chordae tendineae. J Thorac Cardiovasc Surg **47**: 532–538, 1964.
14) Manhas DR, Hessel EA, et al: Repair of mitral incompetence secondary to ruptured chordae tendineae. Circulation **43**: 688–697, 1971.
15) McGoon DC: Repair of mitral insufficiency due to ruptured chordae tendinae. J Thorac Cardiovasc Surg **39**: 357–361, 1960.
16) Piffarre R, Caralps JM, et al: Mitral valve replacement for regurgitation in an infant with Marfan's syndrome. J Thorac Cardiovasc Surg **67**: 233–236, 1974.
17) Rastelli GC, Tsakiris AG, et al: Exercise tolerance and hemodynamic studies after replacement of canine mitral valve with and without preservation of chordae tendineae. Circulation **35**: 34–40, 1967.
18) Ross BA, Fox C, et al: Late results of valvoplasty for mitral regurgitation due to rupture of chordae of the posterior (mural) cusp. J Thorac Cardiovasc Surg **76**: 533–536, 1971.
19) Shore DF, Wong P, et al: Surgical management of ruptured chordae. Thorax **35**: 80–85, 1980.

和文抄録

腱索断裂による僧帽弁閉鎖不全症に対する外科治療

島根医科大学　第1外科
岡本　好史，粟津　篤司，大仏　正隆

島根医科大学　第4内科
二瓶　東洋，盛岡　茂文，森山　勝利

大津赤十字病院　外科
渡　辺　　裕

我々は腱索断裂による僧帽弁閉鎖不全症の18例を経験し，その臨床像と外科治療法に検討を加えた．

男11例，女7例で平均年令49才であった．腱索断裂に特有な急性症状を呈したのは9例で他は腱索断裂の発現時期を判定することはできなかった．

病因別にみると，感染性心内膜炎3例，マルフアン症候群1例，修正大血管転位症1例，原因不明3例，他の10例は組織変性によるものと思われた．

弁形成術後再手術症例を経験し，弁組織，弁下組織が脆弱な場合積極的に人工弁置換術を選ぶべきである．

我々は人工弁置換術の8例にSJM人工弁を使用し，弁および弁下組織を切除しない術式を用い良好な結果を得た．