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ADULT CARDIAC

Is it worth repairing rheumatic mitral valve disease in children? Long-term outcomes of an aggressive approach to rheumatic mitral valve repair compared to replacement in young patients†

Paneer Selvam Krishna Moorthy*, Sivakumar Sivalingam, Jeswant Dillon, Pau Kiew Kong and Mohd Azhari Yakub

Department of Cardiothoracic Surgery, National Heart Institute, Kuala Lumpur, Malaysia

* Corresponding author. Department of Cardiothoracic Surgery, National Heart Institute, 145 Jalan Tun Razak, 50400 Kuala Lumpur, Malaysia. Tel: +60-3-26178505; fax: +60-3-26928418; e-mail: paneer@ijn.com.my; paneer269@gmail.com (P.S. Krishna Moorthy).

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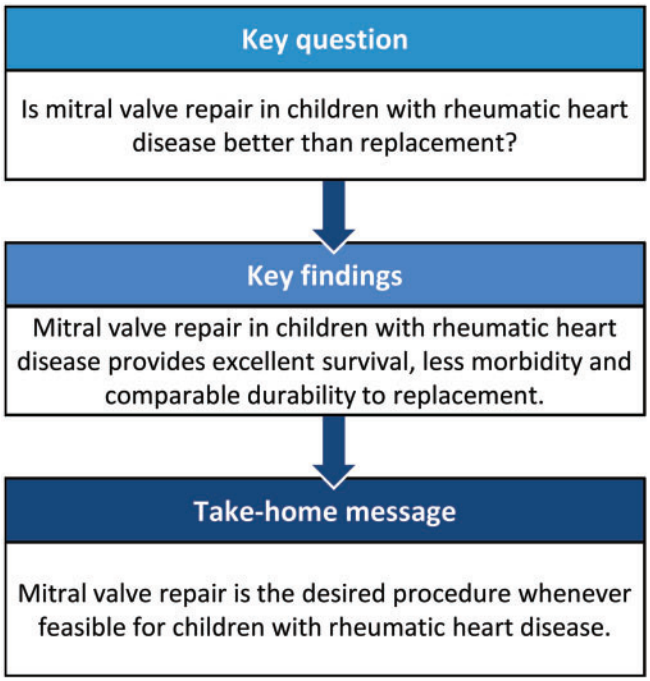

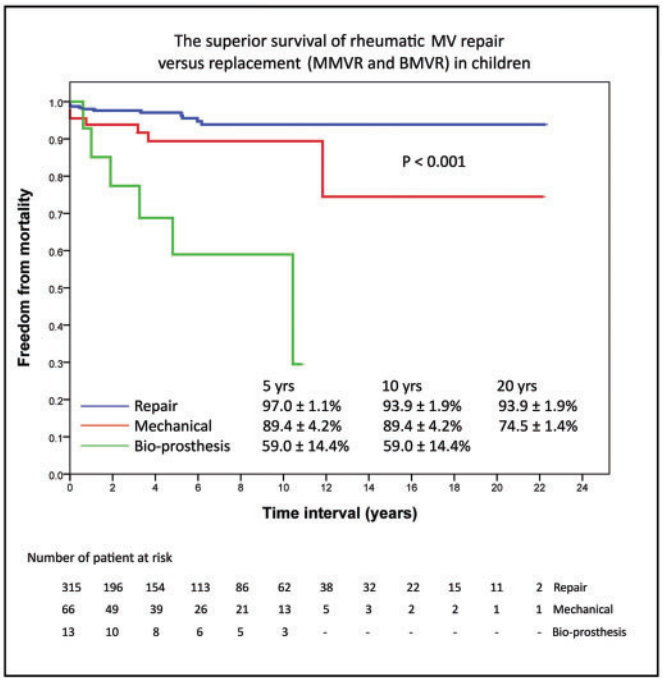

Abstract

OBJECTIVES: Contemporary experience in mitral valve (MV) repair for children with rheumatic heart disease (RHD) is limited, despite the potential advantages of repair over replacement. We reviewed our long-term outcomes of rheumatic MV repair and compared them with the outcomes of MV replacement in children with RHD.

METHODS: This study is a review of 419 children (≤18 years) with RHD who underwent primary isolated MV surgery between 1992 and 2015, which comprised MV repair (336 patients; 80.2%) and MV replacement (83 patients; 19.8%). The replacement group included mechanical MV replacements (MMVRs) ($n = 69$ patients; 16.5%) and bioprosthetic MV replacements ($n = 14$ patients; 3.3%). The mean age with standard deviation at the time of operation was 12.5 ± 3.5 (2–18) years. Mitral regurgitation (MR) was predominant in 390 (93.1%) patients, and 341 (81.4%) patients showed ≥3+ MR. The modified Carpentier reconstructive techniques were used for MV repair.

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RESULTS: Overall early mortality was 1.7% (7 patients). The mean follow-up was 5.6 years (range 0–22.3 years; 94.7% complete). Survival of patients who underwent repair was 93.9% both at 10 and 20 years, which was superior than that of replacement ($P < 0.001$). Freedom from reoperation at 10 and 20 years after MV repair was 81.7% and 72.6%, respectively, compared to 83.2% for MV replacement ($P = 0.580$). Forty patients underwent reoperation after the initial surgery with no operative deaths. Mixed mitral lesion and postoperative residual MR (≥2+) were the predictors for reoperation in the repair group, whereas lower body surface area and usage of bioprosthesis were significant factors for the replacement group. Freedom from thrombotic, embolic and haemorrhagic events at 10 and 20 years for patients with repair was 98.2% compared to 90.1% in patients with replacement and 67.6% for patients with MMVR ($P = 0.004$).

CONCLUSIONS: Twenty-three years of follow-up shows that MV repair is superior to MMVR in children with RHD. Hence, the rheumatic MV should be repaired when technically feasible to maximize the survival and reduce the valve-related morbidity with comparable durability to MMVR.

Keywords: Rheumatic mitral valve disease • Repair • Replacement • Children • Survival • Durability

INTRODUCTION

Rheumatic heart disease (RHD) still poses a major threat to public health in developing nations and is the commonest acquired cardiovascular disease in children [1]. It is estimated that at least 15 million people worldwide are affected, with a prevalence of 30 out of every 1000 children, and is more common among females [1, 2]. Many affected children with RHD require surgery either to repair or replace a valve within 5–10 years of diagnosis, more commonly the mitral valve (MV) [2]. MV repair has evolved to a great extent in all age groups including children with the introduction of standardized and reproducible techniques [3–5]. Reconstruction conserves the ventricular geometry and function, resulting in better long-term survival [4–7]. MV replacement in children has the risk of higher mortality, anticoagulation-related complications, endocarditis and patient–prosthesis size mismatch, as the child grows [8–10].

Many groups have reported excellent results with MV repair for congenital lesions in children [4–7, 11]. The lower frequency of repairs in RHD has been due to complexity of the disease and the unpredictability of long-term outcomes. However, selected centres have reported encouraging results in repairing rheumatic lesions [11–15].

We present our 23 years of experience from a single centre comparing the long-term outcomes, predictors of survival and durability of MV repair and mechanical MV replacement (MMVR) in children with RHD.

MATERIALS AND METHODS

Patients

We present a single-centre review of 419 children (≤18 years) (168 boys and 251 girls) with RHD who underwent primary isolated MV surgery with or without concomitant tricuspid valve repair between 1992 and 2015. This comprised MV repairs (336 patients, 80.2%) and MV replacements (83 patients; 19.8%). MMVR was performed in 69 patients (16.5%) and bioprosthetic MV replacements (BMVR) in 14 patients (3.3%). Patients requiring MV surgery for acute rheumatic carditis and aortic valve surgery were excluded from this analysis.

An aggressive approach was adopted increasing the average feasibility of MV repair in children with RHD at our institution to almost 80.2% (336 of 419 patients). Demographic, intraoperative and perioperative data were recorded prospectively. Study approval was obtained from the ethics committee of the institute.

The mean age with standard deviation at the time of surgery was 12.5 ± 3.5 (2–18) years. Mitral regurgitation (MR) occurred in 390 (93.1%) patients, and 341 (81.4%) of them showed ≥3+ MR. A stenotic lesion was found in 29 patients (6.9%). Pure mitral stenosis was noted in 7 patients (1.7%). Atrial fibrillation was present in 37 (8.8%) patients. Shortness of breath on exertion was the predominant symptom, and 292 (69.7%) patients were in New York Heart Association (NYHA) functional Class II and higher. Patient characteristics, grouped by the type of surgery performed, are listed in Table 1.

Preoperative assessment

As previously described by Yakub *et al.* [11, 15], transthoracic echocardiography was performed to classify the aetiology, pathology and mechanism of MV disease. Severity of MR was divided into 4 grades as in our study [trivial or mild (1+ MR) in 4 patients, moderate (2+ MR) in 49 patients, moderate to severe (3+ MR) in 68 patients and severe (4+ MR) in 273 patients]. Mitral stenosis was graded based on the MV area and mean pressure gradient. Pure MS was severe in 7 patients (mean pressure gradient >10 mmHg) and moderate in 22 patients (mean pressure gradient 5–10 mmHg). All patients weighing above 3 kg had intraoperative transoesophageal echocardiography to analyse the valve before and after the valve repair.

Surgical procedures

Indications for surgery were based on patient's age, symptoms and MV and tricuspid valve lesions. The decision to repair or to replace and the type of the surgical repair were based on the surgeon's preference, patient and valve factors. In general, we have adopted some criteria to help us choose and guide us in repairing the MV. The severity and length of a commissural fusion, pliability of the leaflets and level of calcification will determine the MV repair. The presence of severe commissural fusion >1 cm in length, severely thickened leaflets with calcification preventing proper shaving or peeling and heavy calcification involving more than 1 segment (commissures, leaflets, annulus and subvalvular apparatus) may deter the surgeons from repairing the MV. Details of MV repair techniques are summarized in Table 2, similar to those described by Yakub *et al.* [11, 15]. The mean size of the annuloplasty ring used in MV repair was 27.7 mm (range 16–36 mm). The mean size of the prosthesis used in the MMVR was 28.1 mm (range 19–35 mm) and in BMVR was 29.4 mm (range 25–33 mm).

Table 1: Patient characteristics of the 419 patients undergoing mitral valve surgery

	MV repair	MMVR	BMVR	Overall	*P*-value
Number of patients, *n* (%)	336 (80.2)	69 (16.5)	14 (3.3)	419 (100)	
Female, *n* (%)	203 (60.4)	34 (49.3)	13 (92.9)	250 (59.7)	0.008
Age (years), mean ± SD	12.3 ± 3.4	13.3 ± 3.6	14.9 ± 3.5	12.5 ± 3.5	0.004
BSA (m^2), mean ± SD	1.14 ± 0.30	1.23 ± 0.32	1.25 ± 0.25	1.17 ± 0.31	0.19
NYHA II, III and IV, *n* (%)	237 (70.5)	43 (62.3)	12 (85.7)	292 (69.7)	0.42
LVEF (%), mean ± SD	65.8 ± 10.2	61.8 ± 9.0	59.7 ± 11.9	64.9 ± 10.2	0.009
Indication for surgery, *n* (%)					
Severe MR	315 (93.8)	61 (88.4)	14 (100)	390 (93.1)	0.049
Severe MS	3 (0.9)	4 (5.8)	0 (0)	7 (1.7)	
Mixed MR and MS	18 (5.4)	4 (5.8)	0 (0)	22 (5.3)	
Additional procedure, *n* (%)					
TV repair	106 (31.5)	8 (11.6)	4 (28.6)	118 (28.2)	0.004
Cox-maze	3 (0.9)	0 (0)	0 (0)	3 (0.7)	0.83
Cross-clamp time (min), mean ± SD	78 ± 30	87 ± 45	65 ± 30	79 ± 33	0.038
CPB time (min), mean ± SD	106 ± 36	128 ± 50	100 ± 41	109 ± 40	<0.001
Early mortality, *n* (%)	4 (1.2)	3 (4.3)	0 (0)	7 (1.7)	0.20
Follow-up time (years), mean ± SD	5.6 ± 5.5	5.7 ± 4.8	5.2 ± 3.8	5.6 ± 5.4	0.95

BMVR: bioprosthesis mitral valve replacement; BSA: body surface area; CPB: cardiopulmonary bypass; LVEF: left ventricular ejection fraction; MMVR: mechanical mitral valve replacement; MR: mitral regurgitation; MS: mitral stenosis; MV: mitral valve; NYHA: New York Heart Association; SD: standard deviation; TV: tricuspid valve.

Postoperative management

As previously described by Yakub *et al.* [11, 15], the patients who underwent prosthetic ring annuloplasty or BMVR were routinely administered warfarin for 6 weeks postoperatively, with a target international normalized ratio of 2.0–3.0, continued indefinitely for those who had MMVR and atrial fibrillation. Patients who underwent biodegradable ring annuloplasty were given aspirin (3–5 mg/kg daily) for 3 months. All patients were given oral penicillin as a secondary prophylaxis against rheumatic fever for 10 years or until 40 years of age (whichever is longer) as is currently recommended [16].

Follow-up

The follow-up information was obtained from hospital records, family practice records and directly from the patient or their family via telephone interviews. Follow-up was 94.7% complete, with 22 patients (5.3%) being lost from the follow-up. The mean follow-up was 5.6 years (range 0–22.3 years). All valve-related events are reported in accordance with the revised guidelines published by the '*Ad Hoc* Liaison Committee for Standardizing Definitions for Prosthetic Heart Valve Morbidity' (2008) [17, 18]. Reoperation was performed for the same indications as the initial operation.

Statistical analysis

Data are presented as frequencies or means with standard deviations. A univariable analysis of categorical data was carried out with the χ^2 test or Fisher's exact test. A univariable analysis of continuous variables was carried out with the Student's test. The Cox regression analysis was used to determine the risk factors for survival, reoperation and valve-related events. Variables with *P*-value ≤0.1 for the univariable analysis were subjected to the multivariable analysis. Analysis of survival and freedom from reoperation, anticoagulation-related complications and valve-related events were performed with the Kaplan–Meier estimator. A *P*-value <0.05 was considered significant. IBM SPSS Statistics for Windows, Version 25.0, was used for statistical analysis.

Table 2: Details of mitral valve repair techniques in 336 patients

Mitral valve repair techniques	Number of cases (%)
Ring annuloplasty	311 (92.6)
Rigid, complete ring	88 (26.2)
Semirigid, complete ring	98 (29.2)
Flexible, complete ring	63 (18.8)
Flexible, partial ring	48 (14.3)
Biodegradable	14 (4.2)
Non-ring annuloplasty	20 (6.0)
Leaflet procedure	158 (47.0)
Leaflet resection (triangular/quadrangular/ thinning/peeling/shaving)	94 (28.0)
Leaflet extension or augmentation (anterior:posterior:both)	43 (12.8) 9:30:4
Leaflet plication	21 (6.0)
Chordal procedure	183 (54.5)
Chordal replacement (neochordae)	113 (33.7)
Chordal shortening	20 (6.0)
Chordal transfer	24 (7.1)
Chordal resection	26 (7.7)
Commissurotomy (splitting only)	27 (8.0)
Commissuroplasty (splitting + sliding plasty/figure of '8' suture)	38 (11.3)
Papillary muscle splitting	30 (8.9)

RESULTS

Early hospital outcomes

All patients survived the operation. The patients were treated in the intensive care unit for a period of 1–44 days (median 1 day),

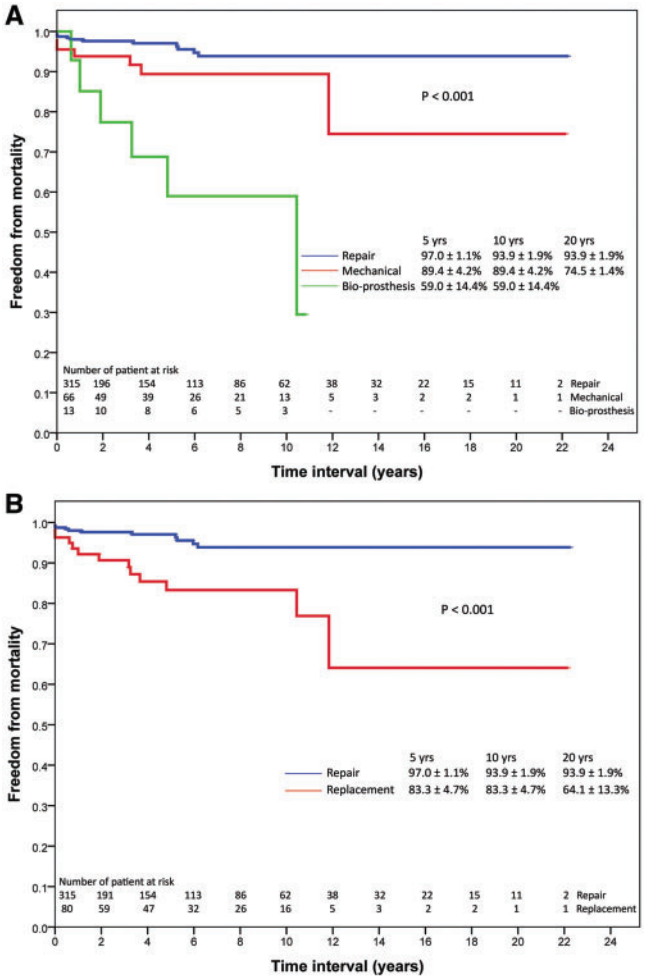

Figure 1: Survival. (**A**) Mitral valve (MV) repair versus mechanical MV replacement versus bioprosthesis MV replacement. (**B**) MV repair versus mechanical MV replacement.

and the mean hospital stay was 10.0 days (range 1–57 days). The 30-day hospital mortality for all 419 patients was 1.7% (7 patients), only 1.2% for patients undergoing repair (4 patients) and 4.3% in the replacement group (3 patients in MMVR and none in BMVR). Persistent low cardiac-output syndrome was responsible for 5 deaths. Other causes of early death included septicaemia ($n = 1$) and severe gastrointestinal haemorrhage ($n = 1$).

Late outcomes

Survival. Survival (including all early and late deaths) at 10 and 20 years following repair was 93.9% compared to MMVR, which was 80.1% and 66.8%, respectively (BMVR at 10 years 59%) ($P < 0.001$; Fig. 1).

The total number of late deaths was 18 patients (4.3%) including 10 cardiac deaths, 3 of non-cardiac cause and 5 patients with unknown cause. There were 8 late deaths following MV repair due to cardiac failure (4), pneumonia (2) and unknown cause (2). There were 4 late deaths following MMVR due to acute valve thrombosis (2), 1 with motor-vehicle accident and 1 with sudden unexpected death. There were 6 late deaths after BMVR: 3 from cardiac failure secondary to severe structural tissue degeneration, 1 from infective endocarditis and 2 from unknown causes. The majority of the patients were in NYHA Class I (92.1%). Most of the survivors in our series either had no residual MR (248 patients; 59.2%) or 1+ MR (trivial or mild MR) (113 patients; 26.9%) recorded at their last follow-up visit. Additionally, many of those who had residual 2+ MR (22 patients; 5.3%) were asymptomatic. All survivors were found to be in sinus rhythm except 22 (5.3%) patients who had atrial fibrillations.

The univariable Cox regression analysis (Table 3) revealed 5 risk factors that were predictive for decreased long-term survival: younger age, impaired left ventricular ejection fraction <50%, mixed MS/MR and pure MS, MMVR and BMVR. However, in the multivariable analysis, only impaired left ventricular ejection fraction <50%, mixed MS/MR and pure MS and BMVR remained statistically significant (Table 3).

Reoperation. Freedom from reoperation at 10 years was worst in the BMVR patients (MV repair 81.7 ± 3.4%, MMVR 83.2 ± 9.2% and BMVR 59.4 ± 16.9%) ($P = 0.015$) (Fig. 2A). However, there was no significant difference in freedom from reoperation at 10 and 20 years between MV repair (81.7 ± 3.4% and 72.6 ± 6.1%) and MMVR (83.2 ± 9.2%) ($P = 0.580$) (Fig. 2B).

Forty patients (9.5%) underwent MV reoperation after the initial procedure [MV repair 33 (9.8%), MMVR 3 (4.3%) and BMVR 4 (28.6%)] with no operative mortality. The mean interval from the initial surgery was 4.8 years (range 0.1–20.1 years). Thirty-three (9.8%) patients after repair underwent redo-mitral surgery (5 patients had re-repair and 28 patients MV replacement). Progression of the disease with associated stenosis and recurrent insufficiency accounted for reoperations in 20 patients (60.6%), whereas technical failures of the initial repair were responsible for the other 5 patients (15.2%) (4 had residual prolapse and 1 had ring dehiscence) including 1 patient who developed haemolysis, requiring a second procedure during the same hospital stay. Three patients (3.0%) had infective endocarditis leading to reoperation. Three patients (4.3%) in the MMVR group had undergone redo MV surgery due to 2 cases of valve thrombosis and 1 case of severe paravalvular leak. Four patients (28.6%) in the BMVR group underwent reoperation for severe structural tissue degeneration (3 patients with severe MS and 1 patient with severe MR). During reoperation, MV replacement was performed in 35 patients (87.5%), and a second MV repair (re-repair) was done in 5 patients (12.5%).

Significant predictors of reoperation in the entire group were smaller body surface area [hazard ratio (HR) 0.04, 95% confidence interval (CI) 0.002–0.68; $P = 0.027$] and BMVR (HR 12.40, 95% CI 1.26–121.83; $P = 0.031$). Further analysis of the MV repair group showed mixed MV lesion and pure MS (HR 2.95, 95% CI 1.03–8.48; $P = 0.045$) and postoperative residual MR (≥ +2) (HR 6.18, 95% CI 1.54–24.85; $P = 0.010$) as significant predictors for reoperation.

Thrombotic, embolic and haemorrhagic events. Freedom from (all early and late) thrombotic, embolic and haemorrhagic events at 5, 10 and 20 years following BMVR were 100%, 100% and not applicable; MV repair were 99.6%, 98.2% and 98.2%; and MMVR were 95.9%, 88.5% and 66.4% (Fig. 3), respectively ($P = 0.005$).

Twenty-three patients in the MMVR group had encountered thrombotic, embolic and haemorrhagic events. Three patients had thrombosed mechanical valves. Ten patients had embolic events: strokes (6) and transient ischaemic attacks (4). Four

Table 3: Univariable and multivariable analyses of risk factors for mortality in 419 patients

Variables	Univariable analysis for mortality			Multivariable analysis for mortality		
	HR	95% CI	*P*-value	HR	95% CI	*P*-value
Female	1.21	0.54–2.75	0.64			
Age (years)	1.20	1.04–1.38	0.014*			
BSA (m^2)	1.14	0.20–6.54	0.88			
LVEF <50%	4.43	1.27–15.39	0.019*	4.34	1.16–16.24	0.029*
Mixed MR/MS and pure MS	3.02	1.03–8.83	0.043*	5.66	1.50–21.38	0.011*
MMVR	2.67	1.05–6.79	0.039*			
BMVR	11.22	4.18–30.12	<0.001*	17.30	5.25–57.01	<0.001*
Concomitant TV surgery	0.97	0.41–2.32	0.95			

* indicates significant of *P*-values.

BMVR: bioprosthesis mitral valve replacement; BSA: body surface area; CI: confidence interval; HR: hazard ratio; LVEF: left ventricular ejection fraction; MMVR: mechanical mitral valve replacement; MR: mitral regurgitation; MS: mitral stenosis; TV: tricuspid valve.

patients had major haemorrhagic events: cardiovascular accident (2), retroperitoneal haemorrhage (1) and gastrointestinal bleeding (1). Six patients had minor haemorrhagic events: haematuria (3), spontaneous bruises (2) and epistaxis (1).

Event-free survival. As described by Remenyi *et al.* [18], we observe similar data on freedom from all late valve-related events (late valve-related death, thrombosis, embolism, haemorrhage, endocarditis and reoperation). At 5, 10 and 20 years, freedom from all late valve-related events was greater in the MV repair group (95.7%, 87.9% and 82.9%, respectively) compared to that of the MMVR group (86.7%, 65.6% and 49.2%, respectively) and the BMVR group (45.5%, 36.4% and not applicable) (Fig. 4) ($P < 0.001$). Significant predictors for valve-related events were mixed mitral lesions and pure MS (HR 3.29, 95% CI 1.10–9.84; $P = 0.033$), MMVR (HR 2.77, 95% CI 1.19–6.45; $P = 0.018$) and BMVR (HR 7.65, 95% CI 2.47–23.67; $P < 0.001$).

Bioprosthetic valve replacement: late outcomes. As described by Remenyi *et al.* [18], we observed similar findings with the BMVR group. All 14 patients were female with complete follow-ups, and 10 patients had major valve-related events (Fig. 4). Six died and 4 had reoperations. None had proved endocarditis or anticoagulant-related complications.

DISCUSSION

The repair of the rheumatic MV is technically more difficult, challenging and complex. MV replacement is not the best option in children due to higher mortality and morbidity in addition to poor compliance with anticoagulation, somatic growth and pregnancy [8–10, 19, 20]. Therefore, MV repair is the desired procedure for children with mitral lesions of all aetiologies [4–7, 11–15].

Over the last 2 decades, our practice has evolved towards a more aggressive strategy of MV repair. This study showed a clear survival advantage and a 20-year survival rate of 93.9 ± 1.9% following MV repair compared to lower rates in MMVR and BMVR for young patients with RHD. As described by Remenyi *et al.* [18], we also found early reoperation in 9.8% of patients who underwent repair, and freedom from reoperation was not significantly different from MMVR group for the duration of the follow-up. In the MMVR group, almost 40% of the patients had a significant thrombotic, embolic or haemorrhagic events within 15 years. This is despite the constant improvement in high profile mechanical valve and the anticoagulation service in the country. In comparison, those who underwent MV repair were almost 98% free from these events leading to a better quality of life.

Although this is the first study with a larger sample size to show improved long-term survival for MV repair compared to MMVR for RHD in the young patients, it is in concordance with the findings of earlier comparative studies with a long-term follow-up [18, 21]. Geldenhuys *et al.* [21] showed a decent freedom from valve-related mortality of 96 ± 3% and 80 ± 11% at 10 years for repairs and replacements, respectively. Remenyi *et al.* [18] also demonstrated a survival advantage in children following MV repair in RHD with survival of 90% compared to 44% following MMVR at 14 years post-surgery. Non-comparative studies in similar young rheumatic populations show equally optimistic results following MV repair [11–15].

The rationale for MMVR rather than MV repair in children is the longer freedom from reoperation. Remenyi *et al.* [18] showed no significant difference in freedom from late operation at 14 years following MV repair compared to MMVR (76% and 73%, respectively; $P = 0.52$). However, the rate of reoperation has been found to be high in young patients after prosthetic valve replacement in some series due to patient–prosthesis size mismatch caused by somatic growth and valve thrombosis [10, 18, 22, 23]. Brown *et al.* [10] reported a 37% reoperation rate during a 35-year follow-up period caused mainly by patient–prosthesis size mismatch due to the use of small prosthesis size during the first operation. Alsoufi *et al.* [22] also reported a 20-year survival and freedom from reoperation of only 74% and 49%, respectively, with half of the patients having reoperation within 20 years of their first MV replacement (mean age at first surgery 11.4 years). Many non-comparative studies have reported freedom from late reoperation for MV repair ranging from 78% to 93% at 10 years [11–15], which are similar to our results.

Patterns of mitral lesions in RHD among children favour valve repair [19, 20]. Marcus *et al.* [19] found that pure regurgitation was the most common lesion in the first and second decades, whereas the relative prevalence of pure stenosis and severe tissue lesions increased with age. Duran *et al.* [20] found that the repair rate in rheumatic mitral disease was related to the patient's age (76.6% < 20 years, 59.1% > 20 < 40 and

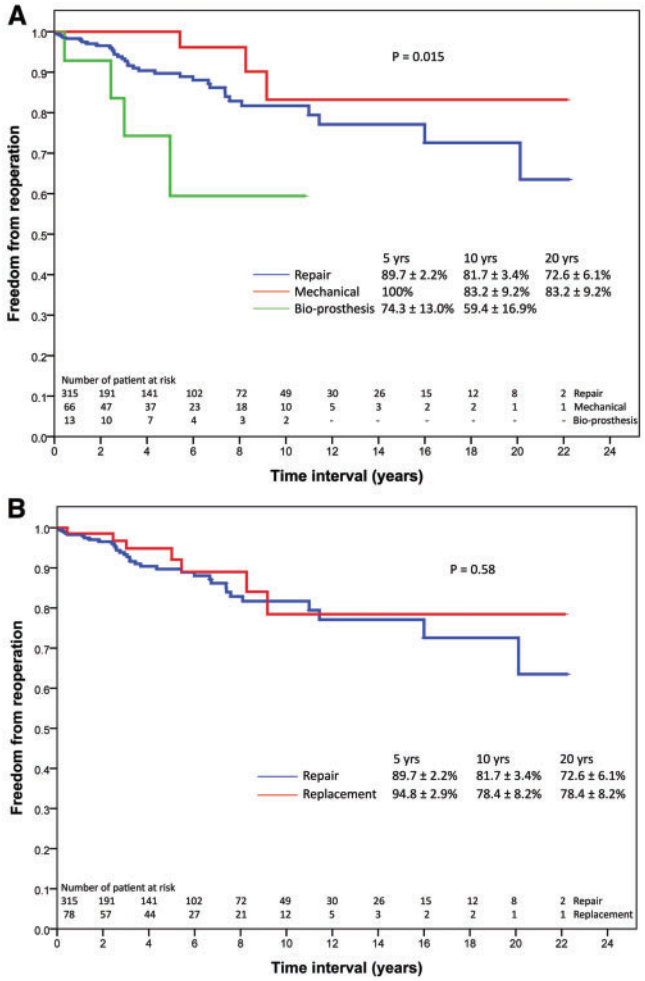

Figure 2: Freedom from reoperation. (**A**) Mitral valve (MV) repair versus mechanical MV replacement versus bioprosthesis MV replacement. (**B**) MV repair versus mechanical MV replacement.

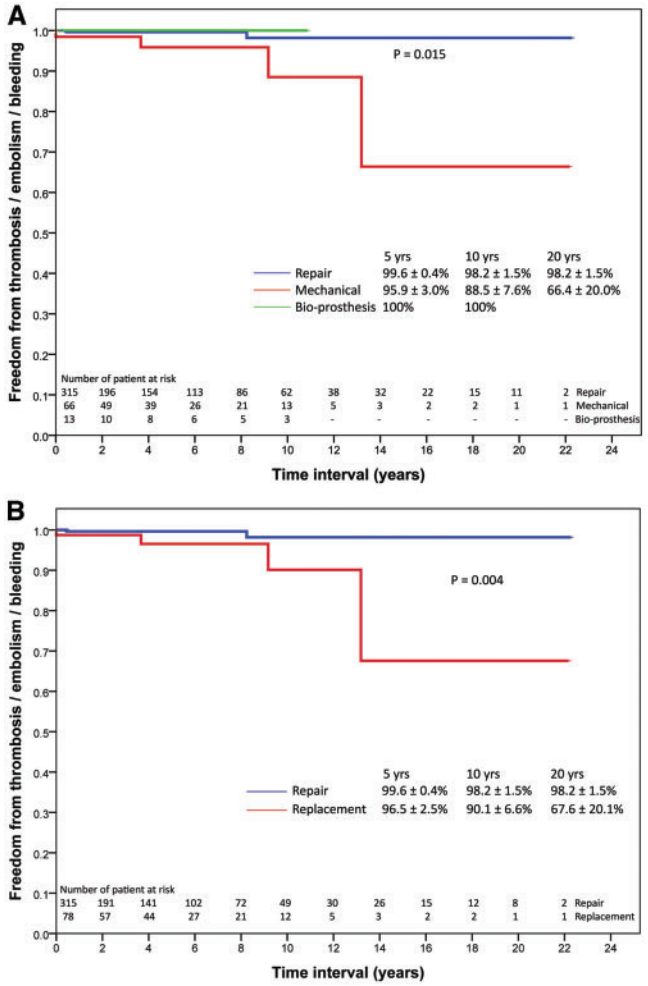

Figure 3: Freedom from valve thrombosis, embolism and bleeding. (**A**) Mitral valve (MV) repair versus mechanical MV replacement versus bioprosthesis MV replacement. (**B**) MV repair versus mechanical MV replacement.

33.8% > 40 years). These similar patterns of disease were seen in our study as well, which documented predominant MR in 93.1% of the patients.

Improvement in repair techniques in the last 3 decades have resulted in better outcomes in treating rheumatic lesions as reported by several studies [11–15, 18, 21]. Prosthetic ring annuloplasty has become a mainstay of MV repair. Conversely, long-term studies [11, 13–15] have successively demonstrated the importance of correcting the annular dilatation and deformity in RHD. Stabilizing the repair using rigid or semirigid ring in its complete form is our preferred method, if the calculated annular size is ≥26 mm. For any size smaller than this, we tend to use bands or biodegradable rings. Moreover, the so-called non-classical techniques (leaflet procedures, which included extensive commissurotomy, thinning, shaving, plication and leaflet augmentation with pericardial patch and neochordae implantation) have further extended the feasibility for rheumatic MV repair with many experienced centres reporting good long-term results [11, 14, 15, 24–28]. Dillon *et al.* [26] demonstrated the leaflet augmentation technique and a good mid-term outcome. Traditionally, leaflet prolapse was repaired by shortening or transferring of the elongated chordae, but recently we favoured chordal replacement using polytetrafluoroethylene material, which has good outcomes in children [27, 28]. Minami *et al.* [27] showed echocardiographic findings of a biological adaptation of children who had chordal replacement with polytetrafluoroethylene, resulting in compensatory excessive growth of MV leaflet and papillary muscle.

The strategy for full resection of all fibrotic tissue followed by valve reconstruction with autologous or heterologous material may increase the repair rate and better durability as residual fibrotic tissue strongly correlates with disease progression leading to repair failure over time [5, 11, 15, 24, 25].

Intraoperative assessment of repaired MV using transoesophageal echocardiography is crucial. It is important not to accept both residual MR > 1+ and residual eccentric MR to improve the outcome as these are the consistent predictors for reoperation [11, 14, 15, 21]. We strive to achieve a leaflet coaptation length of more than 5 mm to ensure long-term durability of the repair.

We identified mixed MV disease with commissural fusion and early residual MR >2+ as the predictors of reoperation in the MV repair group, which were also noted in other studies [11, 15, 21]. The early peak in reoperation may be attributed to suboptimal repair and late valve failures, which have been attributed to recurrence and progression of the inflammatory process in rheumatics [11, 14, 15].

Our experience with bioprosthetic valves in the mitral position in children with RHD is limited. However, 10 of 14 patients in this series had a significant valve-related event within 5 years,

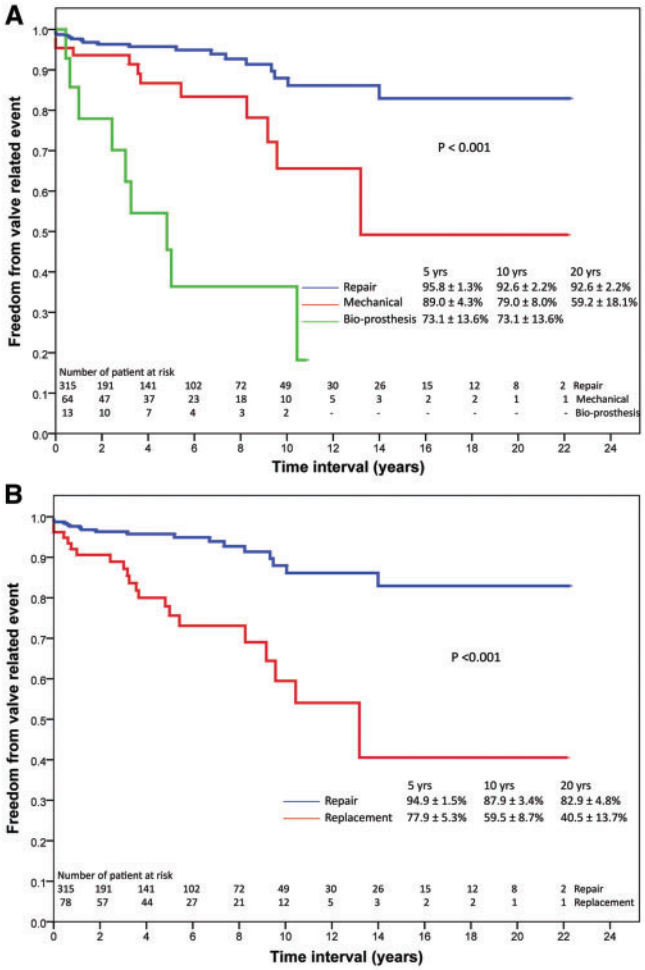

Figure 4: Freedom from valve-related events. (**A**) Mitral valve (MV) repair versus mechanical MV replacement versus bioprosthesis MV replacement. (**B**) MV repair versus mechanical MV replacement.

with survival and freedom from reoperation at 5 years being 59 ± 14.4% and 74.3 ± 13.0%, respectively. Others have also noted a relative high mortality and morbidity associated with BMVR in children [18, 22, 23]. This experience indicates that BMVR in children should be avoided.

Limitations

This non-randomized study is susceptible to referral, procedural and institutional biases. Not all MVs are reparable, and those who underwent replacement likely had less favourable anatomy. There are variations in MV repair based on the surgeon's experience with different techniques and level of acceptance on the results of repair.

CONCLUSIONS

MV repair in children with RHD is feasible and offers an excellent survival advantage and a greater freedom from valve-related morbidity with comparable durability to replacement. Modifications of standard repair techniques, adherence to the importance of good leaflet coaptation and avoidance of residual MR have improved the long-term results. BMVR should be avoided in the mitral position in young patients.

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