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Surgical treatment of aortic regurgitation accompanying ventricular septal defect and long term results

Ventriküler septal defekt ve aort yetersizliğinde cerrahi tedavi ve uzun dönem sonuçları

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Background: We present the long-term results of patients operated for aortic regurgitation accompanying ventricular septal defect (VSD).

Methods: Out of 314 patients operated for VSD between 1985-2004, 9 patients (2.86%) (7 males, 2 females; mean age 21.44±12.9 years; range 8 to 45 years) had VSD associated with aortic regurgitation. In six patients VSD was subaortic whereas in three patients it was located in the perimembranous septum. Two patients had history of infective endocarditis. VSD was repaired via transaortic route in four cases. In three of the remaining five patients, the defect was reached and repaired through right atrial incision, in one patient through right ventricular incision and in one patient through both right atrial and right ventricular incisions. In eight patients VSD was repaired with dacron patch and in one patient with native inverted insitu right coronary cusp tissue. In three cases with moderate aortic regurgitation, resuspension was performed to relieve regurgitation. Aortic valve replacement was performed in six patients with aortic valves unsuitable for repair.

Results: Mean follow-up of patients was 62.5±40 (7-120) months. There was no early mortality. Atrioventricular block was not observed in any of patients. Significant aortic regurgitation was not detected upon postoperative echocardiographic controls of three patients who underwent aortic valve repair. Postoperative mean functional capacity showed significant improvement in accordance to preoperative values (p=0.016). On echocardiographic measurements, a statistically significant improvement was detected between preoperative and postoperative mean left ventricular end-systolic and end-diastolic diameters (p=0.034, <0.0001).

Conclusion: Long-term results of patients operated for ventricular septal defect associated with aortic regurgitation are good and left ventricular functions and funtional capacity show significant improvement after both aortic valve repair and replacement.

Key words: Aortic valve insufficiency/surgery; heart septal defects, ventricular/surgery.

Amaç: Ventriküler septal defekt ve aort yetersizliği bulunan hastaların cerrahi tedavisinin uzun dönem sonuçları incelendi.

Çalışma planı: 1985-2004 yılları arasında ventriküler septal defekt ile birlikte aort yetersizliği bulunan dokuz hastaya (%2.86) (7 erkek, 2 kadın; ort. yaş 21.44±12.9; dağılım 8-45) girişim yapıldı. Hastaların altısında ventriküler septal defekt subaortik yerleşim gösterirken, üçünde perimembranözdü. İki olguda geçirilmiş endokardit öyküsü olduğu saptandı. Ventriküler septal defekt, uygun olan dört olguda transaortik, geri kalan beş olgunun üçünde sağ atriyal, bir olguda sağ ventriküler, bir olguda ise hem sağ atriyal hem de sağ ventriküler kesi ile onarıldı. Hastaların sekizinde ventriküler septal defekt Dacron ile kapatılırken, birinde sağ koroner kusp inverte edilerek yama olarak kullanıldı. Orta dererecede aort yetersizliği olan üç olguya resüspansiyon uygulanarak yetersizlik giderildi. Onarıma uygun olmayan altı hastada kapak replasmanı yapıldı.

Bulgular: Çalışmaya alınan hastalar ortalama 62.5±40 (7-120) ay izlendi. Hiçbir hastada erken ve geç mortalite görülmedi ve atriyoventriküler blok gözlenmedi. Aort kapağına onarım uygulanan üç olgunun ameliyat sonrası yapılan ekokardiyografik kontrollerinde anlamlı bir kaçak saptanmadı. Ameliyat sonrası ortalama efor kapasiteleri ameliyat öncesi döneme oranla düzelme gösterdi (p=0.016). Ekokardiyografik ölçümlerde ortalama sol ventrikül sistol sonu çapı ve diyastol sonu çapları ameliyat sonrasında anlamlı derecede düzeldi (p=0.034, p<0.0001).

Sonuç: Ventriküler septel defektle birlikte aort regürjitasyonu nedeniyle ameliyat edilen hastalarda, aort kapağı tamiri ve replasmanı sonrası uzun dönemde sol ventrikül fonksiyonları ve efor kapasitesinde belirgin düzelme sağlanmaktadır.

Anahtar sözcükler: Aort yetersizliği/cerrahi; ventriküler septal defekt/cerrahi.

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Aortic regurgitation (AR) accompanying ventricular septal defects (VSD) is an anatomic anomaly which usually results from prolapsus of right coronary cusp (RCC), noncoronary cusp (NCC) or both. Its incidence is between 4.5-11%.[1] It is more frequent in Japan and China. Garamella et al.[2] and Starr et al.[3] suggested the surgical treatment for the first time in 1960. Afterwards, techniques including plication of prolapsed leaflet by pledgeted mattress suture were introduced by Trusler et al.[4] and Spencer et al.[5] separately in 1973. Chauvaud et al.[6] and Carpentier[7] introduced triangular valvular resection and reported good results. Yacoub et al.[8] repaired prolapsed RCC transaortically with some pledgeted sutures passed through rim of VSD, which plicated prolapsed sinus valsalva meanwhile reinforcing weak sinus base with the ventricular crest. However, in old patients with advanced prolapsus and retraction developed by time, in patients with aortic valves showing structural deformity or destruction secondary to infective endocarditis aortic valve replacement may be obligatory.

PATIENTS AND METHODS

Between 1985-2004 a total of 314 patients underwent surgical intervention with diagnosis of congenital VSD in our clinics. In 9 of these patients (2.86%) VSD was associated with aortic regurgitation. Seven of the patients were male and mean age was 21.44±12.9 (8-45) years. Mean functional capacity of patients according to NYHA (New York Heart Association) was 2.75±0.7. In six patients ventricular septal defect was subaortic whereas in three patients it was located in perimembranous septum. In all patients diagnosis was done by transthoracic echocardiography. Cardiac catheterization was performed in addition to echocardiography in four patients. Mean pulmonary artery pressure of patients was 33.5±27.9 mm Hg. Two patients had history of infective endocarditis. In one of these patients severe mitral regurgitation developed as a result of endocarditis. Mean diameter of VSD was 1.17±0.52 cm whereas mean shunt ratio of VSD was 1.65±0.42. Mean grade of aortic regurgitation was 3.25±0.46. Mean preoperative left ventricular end-systolic diameter was 3.82±0.73 cm and mean end-diastolic diameter was 5.9±0.68 cm.

Surgical tecnique. All patients were operated on an elective base. Operations were performed through median sternotomy under general anesthesia. Aortic cannulation was performed at distal ascending aorta and bicaval venous cannulation was performed. Vena cavae were encircled by silastic tapes. Venting cannula was inserted through right superior pulmonary vein. All operations were performed under moderate (28 °C) hypothermia. After aortic cross clamping, myocardial protection was performed through coronary artery ostiums directly after oblique aortotomy using St Thomas II solution in three patients, by continuous isothermic retrograde blood cardioplegia in five patients and by using combined antegrade/retrograde blood cardioplegia in one patient. Following aortotomy exploration was done to determine if aortic valve was suitable for reconstruction and to decide whether it is suitable to repair VSD via aortotomy. In four patients VSD was repaired through aortotomy. In three of remaining five patients, defect was reached and repaired through right atrial incision, in one patient through right ventricular incision and in one patient through both right atrial and right ventricular incisions. In all patients ventricular septal defect was repaired with dacron patch using pledgeted 3/0 dexon sutures. In one patient alternativly VSD closure with native inverted insitu RCC tissue. Two of three cases with moderate aortic regurgitation, resuspension of prolapsed RCC using technique of Trusler was performed to relieve regurgitation. In one patient resuspension of both RCC and NCC was performed in one patient by using the same method. Aortic valve replacement was performed in five patients with aortic valves unsuitable for repair. In one of two patients with history of previous endocarditis, severe mitral regurgitation was present and mitral valve replacement was performed concomitantly (Table 1).

Statistical analysis. Data are presented as mean ± standart deviation. Preoperative and postoperative left ventricular diameters were analysed using Paired t-test, preoperative and postoperative NYHA functional class was analysed using Wilkoxon signed ranks test. Values below 0.05 were accepted to be statistically significant.

Follow-up. The mean follow-up period was 62.5±40 (7-120) months. Routine control data of all patients were collected retrospectively and prospectively. Routine cardiac examination and echocardiography were performed and NYHA functional capacity was noted. Left ventricular end-systolic and end-diastolic diameters, ejection fraction, degree of aortic regurgitation in patients with aortic valve repair, function of prosthetic valve in patients with aortic valve replacement and state of septal patch were asessed.

RESULTS

There was no early mortality. Atrioventricular block was not observed in any of the patients. Atelectasis developed in two patients and ventricular premature beats treated with medical therapy was observed in one patient. Mean cross clamp time was 119±33.3 minutes and mean cardiopulmonary bypass time was 147.2±36.6 minutes. Mean stay in intensive care unit was 2.33±0.51 days whereas mean hospital stay was

Table 1. Patient's data

Age	Sex	VSD diameter (cm)	Shunt ratio	Location	AI (°)	Operation
8	Male	1. 5	1. 5	Subaortic	3	Aortic repair. + VSD closure
11	Male	0. 7	2. 5	Subaortic	4	AVR + VSD closure
13	Female	2	2	Perimemb.	3	AVR + VSD closure
17	Male	1. 5	1. 5	Subaortic	3	Aort repair. + VSD closure
19	Female	1. 5	1. 5	Subaortic	3	Aort repair. + VSD closure
21	Male	0. 5	1. 2	Perimemb.	4	AVR + VSD closure
41	Male	1	1. 7	Perimemb.	3	AVR + VSD closure
45	Male	0.7	1.3	Subaortic	3	AVR + MVR + VSD closure
18	Male	1.0	1.5	Subaortic	3	AVR + VSD closure

AI: Aortic insufficiency; AVR; Aortic valve replacement; MVR: Mitral valve replacement; VSD: Ventricular septal defect.

10.16±3.54 days. Antiaggregant and anticoagulant treatment was prescribed to patients with aortic valve replacement and prothrombin time was kept around 1.5-2 times of normal value. Significant aortic regurgitation was not detected upon postoperative echocardiographic controls of three patients who underwent aortic valve repair. Ventricular septal patch was intact in all patients. There was no late mortality. Complications related to valve replacement were not detected in our series. On postoperative echocardiographic examination, mean left ventricular end-systolic diameter was regressed to 3.17±0.55 cm (p=0.034) and end-diastolic diameter to 5±0.47 cm (p<0.0001) which was statistically significant. Mean postoperative functional capacity was improved to1.25±0.35 that was statistically significant (p=0.016).

DISCUSSION

Pathophysiology of aortic regurgitation associated with VSD is well known. Since aortic regurgitation worsens with time, timing of surgical intervention and choice of treatment strategy is important. In patients with mild to moderate aortic regurgitation closure of ventricular defect is not enough. Due to progression of aortic regurgitation, reoperation may be necessary in a significant number of patients.[9] Aortic repair becomes more difficult in patients whose operations are postponed till adulthood. For this reason, aortic valve should be assessed intraoperatively in all patients with suspicion of aortic regurgitation. In these patients, valve repair should be the first choice of treatment which is not always successfull. Trusler performed the plication of prolapsed leaflet by fixing it to aortic wall at the site of commisure with pledgeted suture.[4] However it was reported that in patients with thin and weak aortic cusps, after plications at commisural site significant residual regurgitation and recurrence occurs resulting from opening and closure.[10] For this reason Kalangos et al.[11] described the technique of plication at free margin of leaflet using a thin pericardial strip. They suggest that this technique will provide balance to the stress at commisural site and incompetence will be less at long term. However Tirone David proposed that growth of leaflets in children may be prevented by this method. Despite of different results of aortic valvuloplasty in different centers long term results are reported to be satisfactory.[12-14] According to Trusler,[15] Structural anomalies related to more than one commissures, bicuspid aortic valve thin and fenestrated leaflets are factors that affect the result of aortic valvuloplasty negatively. In our study, the suture technique that Trusler described was used in all three patients in whom repair was performed. On long term follow-up, we didn't observe significant regurgitation in any of these patients and reoperation was not necessary. We believe that, in patients with mild to moderate aortic regurgitation without any structural anomaly of leaflet, this simple technique is acceptable. Patients with VSD associated with moderate degree aortic regurgitation should be operated on before reaching adulthood. In adult patients, aortic valvuloplasty may not be successfull because the structural changes of valve will increase with age.[10,15] In these patients aortic valve replacement can be performed safely. Atay et al.[16] performed aortic valve replacement in 4 of their six patients and related this to the older age of their patients at the time of operation. Infective endocarditis should be kept in mind in this group of patients. In our opinion, in patients with a small VSD and mild aortic regurgitation, prophylacsis for infective endocarditis should be considered seriously, because repair of valve will become more difficult after endocarditis. In two of our patients (25%), severe destruction of cusps secondary to infective endocarditis was present and aortic valve had to be replaced in both. In the remaining three patients in whom aortic valve was replaced, severe retraction and substrate loss were present in more than one leaflet. In all of these cases, aortic repair was tried first and since it was unsuccessfull valve was replaced. On long term follow-up of these patients no complications related to mechanical valve wereobserved.

In this group of patients, approach and closure technique of VSD are also important. We believe that VSD should be repaired through aortotomy or right atrium in the first place. VSD should be repaired using a patch. In four of patients for whom aortic valve replacement was performed, VSD was repaired via aortotomy. None of our patients presented with recurrent VSD on follow-up.

If aortic valve repair is not successful, aortic valve should be replaced without hesitation. On long-term follow-up, left ventricular functions and funtional capacity show significant improvement after aortic valve repair and replacement.

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