

Early Surgical Treatment of Ventricular Septal Rupture in Acute Myocardial Infarction

Murat DEMİRTAŞ MD, Fikri YAPICI MD, Hacı AKAR MD, Mehmet KAPLAN MD, Cem ALHAN MD, Hüseyin TOKLU MD, Sabri DAĞSALI MD, Ergin EREN MD, Azmi ÖZLER MD
Siyami Ersek Thoracic and Cardiovascular Surgery Center, İstanbul - Turkey

AKUT MİYOKARD İNFARKTÜSÜNE BAĞLI VENTRİKÜLER SEPTUM RÜPTÜRÜNÜN ERKEN CERRAHİ TEDAVİSİ

Nisan 1990, Haziran 1995 tarihleri arasında, 8 hasta, enfarktüse bağlı ventriküler septum rüptürü nedeniyle erken dönemde opere edildi. Ortalama yaş $65,4 \pm 5$ (57-72 yaşları arası) idi. Olguların % 50'si erkekti. Bir hasta daha önce anterolateral ve inferiyör miyokard enfarktüsü geçirmişti ve apikal anevrizma oluşumu gösteriyordu. Tüm hastalara transtorasik ekokardiyografi, kardiyak kateterizasyon ve koroner anjiyografi yapıldı. Hastaların akut miyokard enfarktüsü tanısıyla hastaneye kabullerinden ameliyata alınmalarına dek geçen süre 36 ile 288 saat (ortalama $101,5 \pm 74,2$ saat; $4,2 \pm 3$ gün) arasında değişiyordu. Interventriküler septum rüptürünün klinik olarak ilk kez tanınması ile cerrahi girişim arasında ise ortalama $39,5 \pm 17$ saat (16 ile 72 saat arası) vardı. Rüptür 5 (% 62,5) olguda anterior, 3 (% 37,5) olguda posteriyör yerleşmişti. Cerrahi öncesi 5 hastada, ameliyat sonrası tüm olgularda IABP kullanıldı. Operasyon öncesi 3 vakada kardiyogen şok mevcuttu ve bunlardan ikisi kardiyopulmoner dolaşımdan ayrılamadı. Dört (% 50) olguya aynı zamanda aortokoroner bypass da yapıldı. Genel hastane mortalitesi % 37,5 oldu, $30 \pm 18,4$ aylık (7 ile 60 ay arası) izleme döneminde de 1 (% 20) olgu reziduel şant nedeniyle tekrar opere edildi. Geç dönemde kaybedilen hasta olmadı.

İnfarktüse bağlı ventrikül septumu rüptürünün erken cerrahi tedavisi, hastane mortalitesi yüksek olmakla birlikte, uzun dönemde kabul edilebilir bir sürvi sağladığından, tercih edilecek tedavi yöntemi olmalıdır. Cerrahi taminin geciktirilmesi erken mortaliteyi iyileştirebilir fakat bu yaklaşım multiorgan yetersizliğinin gelişmesine ve acil cerrahi tedavi ile kurtarılacak olguların kaybedilmesine yol açabilir.

Anahtar kelimeler: Miyokard enfarktüsü, septum rüptürü, erken cerrahi, kardiyogen şok

Rupture of the interventricular septum is a serious mechanical complication of acute myocardial infarction described first by Latham in 1846 at autopsy (1). Infarct-related septal rupture is estimated to complicate 1 - 3 % of acute myocardial infarctions and it produces approximately 5 % of periinfarction deaths (2).

Surgical intervention timing remains unresolved and clinicians face a difficult therapeutic dilemma (3). After the first successful repair of postinfarction ventricular septal rupture by Cooley and colleagues in 1957, the current operative mortality rate ranges from 25 % to 51 % (5), and is worse in patients with cardiogenic shock and in those with a rupture located inferiorly. Early repair of septal rupture carries a mortality of 40 % to 50 %, compared with a mortality of less than 10 % in patients undergoing operations more than 3 weeks after infarction. But only about 15 % of patients can be controlled by conventional medical treatment for the period of 3 to 6 weeks (6). Contrarily, Daggett et al, (7) published a report in 1977 on a series of 43 patients describing increased survival after early surgical repair of the ruptured septum and they attributed this result to improved myocardial preservation and surgical technique.

Here we present our experience of early surgical treatment of ventricular septal rupture in acute myocardial infarction on a limited number of patients.

MATERIAL and METHODS

Patients

Between April 1990 and June 1995, 8 consecutive patients underwent surgical repair of a postinfarction ventricular septal rupture at Siyami Ersek Cardiothoracic Center, İstanbul.

Timing of operation

The time period between the admission to the hospital with the diagnosis of acute myocardial infarction and the operation ranged from 36 to 288 hours, with an average of $101,5 \pm 74,2$ hours ($4,2 \pm 3$ days). The time interval from the first clinical manifestation of interventricular septal rupture to surgery was $39,5 \pm 17$ hours (range 16 to 72 hours).

Therapeutic and diagnostic procedures

Swan-Ganz catheterization was performed and inotropic support was used in all patients. IABP was inserted in 5 patients. All patients underwent bedside transthoracic echocardiography, cardiac catheterization and angiography.

Operative technique

All patients were operated on with standard cardiopulmo-

Received June 30, revision accepted september 28, 1995
Adress of correspondance: Dr. M. Murat Demirtaş, Ahmet Çelebi Mah., Sümbülzade Sok., 20/3, 81160 Üsküdar - İstanbul-Turkey
Tel. : (90) 216 342 25 67 Fax : (90) 212 512 02 78

nary bypass, with the use of moderate (25-28° C) systemic hypothermia and topical saline slush. Myocardial protection was achieved with intermittent cold crystalloid antegrade and retrograde cardioplegia. The circuit was routinely equipped with a membrane oxygenator and a centrifugal pump.

The repair was carried out with the exposure of interventricular septal rupture site by left ventriculotomy performed through the infarcted area. A limited excision of the most friable edges of the defect, completely covering the left side of the septal wall by a Dacron patch with interrupted horizontal mattress sutures through viable septal muscle, and closure of the left ventriculotomy incision with two Teflon felt strips were the following steps of the repair. One patient received a free posterior wall patch (8). De Vega annuloplasty for tricuspid valve was carried out in 1 patient and aneurysmectomy in 2 patients. Coronary artery bypass grafting was performed in 4 cases.

RESULTS

The operative mortality is 37.5 % with 3 patients. Three patients received IABP postoperatively, thus IABP was used in all patients. Two of the 3 cases who presented with cardiogenic shock preoperatively were lost because of low cardiac output state and the inability to end cardiopulmonary bypass. In one of these cases it was not possible to repair the septal defect accurately because the whole septum was thin walled, friable and necrotic. Another patient died because of uncontrollable surgical bleeding from the suture lines of the posterior wall patch.

One case who presented with residual shunt four months later was reoperated on in another cardiac center. She is now alive with a negligible left-to-right shunt without congestive heart failure.

The duration of the follow-up period was 30±18.4 months (2.5±1.5 years) with a range of 7 to 60 months. No death has occurred during this period and the five survivors are in NYHA class II.

The small number of our patients does not permit an accurate risk factor analysis, but the overall early mortality and late results of this series do not differ greatly from that of the other series in the literature (Table 1).

DISCUSSION

Rupture of the interventricular septum occurs most frequently during the first week after infarction (2), and tends to develop in patients experiencing their first transmural infarction in whom collateral flow to the septum is limited. Twenty to 30 percent of the ruptures develop as early as the first 24 hours after the onset of initial symptoms of infarction (9).

Rupture of the interventricular septum produces an abrupt change in clinical status characterized by cardiogenic shock and/or heart failure. Rapid hemodynamic deterioration develops in approximately one-half of the patients (6).

Of the patients with septal rupture, 50 % die within the first week, and 85 % die within 2 months without surgical intervention (10). In 1960's, mortality with medical treatment alone was as high as 87 % within 2 months (11,12), and 93 % of patients were lost within 1 year (2). The current operative mortality rate is worse in patients treated early after infarction and in

Table 1. Reported series of surgical treatment of postinfarct ventricular septal rupture

	Pts (n)	Site of Rupture		Early Mty	Survival (y)			Residual Sht (%)	MTI between SR to op (days)
		Anterior	Posterior		1	5	10		
Held (3) 1978-85	37	52.5 %	47.5 %	51.3%	?	?	?	?	33
Skillington (5) 1973-88	101	56.4 %	43.5 %	20.8 %	71	71	50	22	9
Loisance (11) 1973-89	66	57 %	43 %	45 %	56	44	26	7.5	7
Jones (12) 1970-85	60	60 %	40 %	38 %	47	50	?	?	11
Blanche (13) 1980-90	22	50 %	50 %	36 %	59	47	?	?	6.6
Muehrcke (20) 1971-91	75		withCABG withoutCABG	21 % 26 %		72 30	41 0		
Komeda (21) 1980-89	31	48.4 %	51.6 %	10 %	?	?	?	?	?
Casella (22) 1984-87	16	56 %	44 %	44 %	?	?	?	?	1-30
Kallela (23) 1974-88	13	46 %	54 %	31 %			38.5		
SEC 1990-95	8	62.5 %	37.5 %	37.5 %	62.5	62.5		20	16-72 h

Post:Patients; Mty: mortality; y:year; Sht:shunt; MTI:mean time interval; SR: septal rupture; h:hours op: operation; CABG:coronary artery bypass grafting; SEC: Siyami Ersek Cardiovascular Center

those with cardiogenic shock and in whom rupture is located inferiorly. Age over 65 years appears to be a significant risk factor (5).

Blanche and his colleagues (13) report that patients who have an IABP placed before operation have a better chance for survival than those in whom IABP has not been used; in addition, survivors have a longer time interval between infarction and operation than nonsurvivors. According to Blanche et al (13) risk factors influencing early mortality are diabetes, elevated preoperative right atrial pressure, absence of IABP and time from myocardial infarction to operation; risk factors influencing long-term survival are diabetes, elevated right atrial pressure and extent of coronary artery disease.

Radford and associates (2) reported in 1981 that their treatment of patients with ventricular septal rupture consisted of prompt insertion of IABP if cardiogenic shock was present; cardiac catheterization, left ventriculography and coronary angiography within 6 hours, and surgical repair carried out within the next 12 hours even if IABP had produced apparent hemodynamic stability because this stability was usually temporary.

The general policy is to operate on virtually all patients who are referred. If they have a ventricular septal defect, they need an operation (5).

According to Loisançe et al, (11) the introduction of blood cardioplegia and warm reperfusion techniques lead to improved postoperative left ventricular function, even if there is a rather small number of patients with coronary lesions that can be bypassed.

Seguin and collaborators (14) say that earlier operation may lead to operative complications mainly because of the tissue fragility of the recently necrosed myocardium. They report that these surgical complications may be massive perioperative bleeding through the ventriculotomy or postoperative residual ventricular septal defect, because of the inadequate implantation of the patch. For this reason, Seguin et al (14) propose the reinforcement of the recently necrosed myocardial tissue by the application of a fibrin sealant.

Delay of repair results in an improved early mortality but this approach may result in the development of multiorgan failure and the death of patients who

could have been saved by emergency closure of the defect (12).

Davies et al (10) believe that because of doubt about the benefit of combining ventricular septal defect repair and coronary artery bypass grafting, critically ill patients should be spared time-consuming and potentially dangerous invasive investigations whenever possible in favor of noninvasive confirmation of the diagnosis, followed by urgent surgical repair of the defect alone.

Two-dimensional echocardiography and Doppler color flow imaging are very useful in the diagnosis and localisation of a postinfarction septal defect. These investigations are 100 % specific in excluding ventricular septal defects (15,16).

Skillington et al (5) report that it is difficult to arrive at a sensible "profit and loss" account for coronary artery bypass grafting associated with acquired ventricular septal defect closure. Coronary angiography is timeconsuming and often poorly tolerated; in addition, longer cross-clamp and cardiopulmonary bypass times must increase the myocardial insult. Skillington's (5) study did not show coronary revascularization to carry significant benefit.

Another point of discussion is how to act against residual defects. Skillington's policy has been to close residual defects when they cause symptoms and signs of heart failure; when the recurrent defect is small and asymptomatic or controlled with minimal diuretic therapy, a conservative policy is entirely reasonable and late spontaneous closure can occur (5).

Acute inferior wall infarction complicated by ventricular septal rupture may be accompanied by tricuspid regurgitation, and patients with postinfarction septal rupture who have right ventricular dysfunction tend to experience cardiogenic shock and have a much worse prognosis for survival than those who do not (17,18). Held et al's data suggest that three factors play role in determining survival of the patients managed surgically: systolic blood pressure, right atrial pressure, and duration of cardiopulmonary bypass. An increase in right atrial pressure correlates with an increase in mortality of these patients and right sided heart failure influences surgical outcome negatively. Thus right ventricular dysfunction as a re-

sult of right ventricular infarct is a strong determinant of eventual outcome (3,12,13).

Pulmonary artery balloon counterpulsation and right ventricular assist devices may be used to ameliorate right ventricular dysfunction but the results have not been encouraging. With low right atrial pressure (<15 mm Hg) and well-maintained systolic blood pressure (>90 mm Hg) surgical outcome is said to be good (3). According to Davies et al's (10) experience, arrhythmias are potentially a significant cause of late mortality in survivors of operations, asymptomatic ventricular ectopy is common and increases the risk of sudden death.

Patient's characteristics, early mortality, late survival, time interval between myocardial infarction and operation, and residual shunt rate of different authors are given in Table 1,

We can conclude that early surgical repair of infarct-related ventricular septal rupture, despite its high mortality, is the treatment of choice because it provides acceptable long-term survival, and delay of repair may result in the development of multiorgan failure leading to death.

REFERENCES

1. Latham PM: Lectures on subjects connected with clinical medicine comprising disease of the heart. London, Longmans, Brown, Green and Longmans, 1846. p. 168-176
2. Radford MJ, Johnson RA, Daggett WM, et al: Ventricular septal rupture: A review of clinical and physiologic features and an analysis of survival. *Circulation* 1981; 64: 545-553
3. Held AC, Cole PL, Lipton B, et al: Rupture of the interventricular septum complicating acute myocardial infarction: A multicenter analysis of clinical findings and outcome. *Am Heart J* 1988; 116: 1330-1336
4. Cooley DA, Belmonte BA, Zeis LB, Schnur S: Surgical repair of ruptured interventricular septum following acute myocardial infarction. *Surgery* 1957; 41: 130-137
5. Skillington PD, Davies RH, Luff AJ, et al: Surgical treatment for infarct-related ventricular septal defects. *J Thorac Cardiovasc Surg* 1990; 99: 798-808
6. Morris DC, Walter PF, Hurst JW: The recognition and treatment of myocardial infarction and its complications. JW Hurst et al (eds). *The Heart*. New York, McGraw-Hill, 1990. p. 1070
7. Daggett WM, Guyton RA, Mundt ED, et al: Surgery for post-myocardial infarct ventricular septal defect. *Ann Surg* 1977; 186: 260-270
8. Yapıcı F, Yerlioğlu E, Bilgen F, et al: Akut miyokard

infarktüsü sonrası ventrikül septumunun posterior bölüm rüptürü: İki olgu bildirisi. *Türk Kardiyol Dern Arş* 1994; 22: 276-279

9. Jones EL, Hatcher CR: Techniques for the surgical treatment of atherosclerotic coronary artery disease and its complications. JW Hurst et al (eds). *The Heart*. New York, McGraw-Hill, 1990. p. 2221-2222
10. Davies RH, Dawkins KD, Skillington PD, et al: Late functional results after surgical closure of acquired ventricular septal defect. *J Thorac Cardiovasc Surg* 1993; 106: 592-598
11. Loisançe DY, Lordez JM, Deleuze PH, Dubois-Rande JL, Lellouche D, Cachera JP: Acute postinfarction septal rupture: Long-term results. *Ann Thorac Surg* 1991; 52: 474-478
12. Jones MT, Schofield PM, Dark JF, et al: Surgical repair of acquired ventricular septal defect. Determinants of early and late outcome. *J Thorac Cardiovasc Surg* 1987; 93: 680-686
13. Blanche C, Khan SS, Matloff JM, et al: Results of early repair of ventricular septal defect after an acute myocardial infarction. *J Thorac Cardiovasc Surg* 1992; 104: 961-965
14. Seguin JR, Frapier JM, Colson, P, Chaptal PA: Fibrin sealant for early repair of acquired ventricular septal defect. *J Thorac Cardiovasc Surg* 1992; 104: 748-751
15. Fortin DF, Sheikh KH, Kisslo J. The utility of echocardiography in the diagnostic strategy of postinfarction ventricular septal rupture: A comparison of two-dimensional echocardiography versus Doppler color flow imaging. *Am Heart J* 1991; 121: 25-32
16. Göksel S, Şaşmaz H, Kural T, Gürkaynak F, Ergin A: Akut miyokard infarktüsü sonrası ventriküler septal rüptür. *T Klin Kardiyoloji* 1990; 3: 73-75
17. Feng WC, Singh AK, Moran JM: Tricuspid regurgitation with postinfarction ventricular septal defect. *Ann Thorac Surg* 1990; 49: 659-660
18. Şenocak H, Ateşal S, Karekelleoğlu Ş, Şahin M, Alp N: Akut miyokard infarktüsü sonucu oluşan ventriküler septal defekt. *T Klin Kardiyoloji* 1992; 5: 190-192
19. Deville C, Fontan F, Chevalier JM, Madonna F, Ebner A, Besse P. Surgery of postinfarction ventricular septal defect: Risk factors for hospital death and long-term results. *Eur J Cardiothorac Surg* 1991; 5: 167-175
20. Muehrcke DD, Daggett WM Jr, Buckley MJ, Akins CW, Hilgenberg AD: Postinfarct ventricular septal defect repair: Effect of coronary artery bypass grafting. *Ann Thorac Surg* 1992; 54: 576-882
21. Komeda M, Fremes SE, David TE: Surgical repair of postinfarction ventricular septal defect. *Circulation* 1990; 82 (supp IV): IV243-247
22. Casella G, Ippolito F, Turinetti B, Magnant B: Rotura postinfartuale del sotto interventricolare, valutazione dei principali parametri clinici e della sopravvivenza postoperatoria. *G Ital Cardiol* 1990; 20: 126-132
23. Kallela I, Salo J, Meminen M, Jarvinen A, Kyosola K: Surgical treatment of ventricular septal defect secondary to myocardial infarction. *Ann Chir Gynaecol* 1990; 79: 31-36