SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES & TECHNOLOGY

TRIVANDRUM - 11

PROJECT REPORT

NAME : D-RADJASSEGAR.

PROGRAMME : D.M. (CARDIOLOGY.)

MONTH AND YEAR OF SUBMISSION : DECEMBER 1993.

CERTIFICATE

I, Dr. D.RADJASSEGAR hereby declare that I have actually ~~performed all the procedures listed~~ carried out the project under report.

Signature D Rajasekhar

Place : TRIVANDRUM

Name in D.RADJASSEGAR
capital letters

Date : 03.11.'93

Forwarded. He has carried out the minimum requirement of procedures / etc.

Signature

Head of the department

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PROJECT REPORTS

~~LIST OF PROCEDURES DONE~~

PROJECT REPORT

TITLE OF THE PROJECT: THROMBOLYTIC THERAPY FOR PROSTHETIC VALVE THROMBOSIS.

NAME ..... D.RADJASSEGAR

PROGRAMME : ..... D.M(CARDIOLOGY)

MONTH & YEAR
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THROMBOLYTIC THERAPY FOR PROSTHETIC VALVE THROMBOSIS

SUMMARY

During 1992-'93, 11 patients (4 males, 7 females) with thrombosed prosthetic cardiac valves were treated with streptokinase on 12 occasions (one patient with prosthetic tricuspid valve had valve thrombosis twice). Their age ranged from 14 to 52 years (median 38.). Two valves were in aortic position 5 were in mitral and 4 were in tricuspid position. Seven were Bjork Shiley prostheses, 3 were Medtronic Hall valves and one was a St.Jude valve. Timing of prosthetic valve thrombosis ranged from 3 months to 11 years after valve replacement surgery. Duration of symptoms due to valve thrombosis ranged from 1-4 months with tricuspid valve thrombosis and 1-14 days with left sided valve thrombosis. Five were in NYHA class II, 6 were in class III and one was in class IV. All patients were evaluated by echo doppler and cine fluoroscopy. Loading dose of streptokinase was 2.5L units in 4 patients and 1 L units in 8 patients. Maintenance infusion was at 1000 units/Kg/hr in 10 patients and 1 L units/hr in 2 patients. Duration of streptokinase infusion ranged from 3 h to 38 h. Thrombolytic therapy was successful (clinical, echo-doppler and cine fluoroscopy) in 11 out of 12 cases (92%). It was unsuccessful in a patient with valve at tricuspid position in whom infusion had to be stopped after 24 h due to gum bleed. One patient developed intracerebral bleed and expired. In conclusion streptokinase therapy is useful for prosthetic heart valve thrombosis.

INTRODUCTION

Prosthetic valve replacement is a standard method of treating patients with hemodynamically significant cardiac valvular lesions. Despite improvements in the quality, durability and hemodynamic characteristics of prosthetic valves, their potential for life threatening complication such as valve thrombosis remains. Thrombotic occlusion of prosthetic valve occurs in 1% to 13% of cases[1], [2] and requires prompt diagnosis and management. Emergency surgery (thrombectomy or valve replacement) has been the conventional treatment for this complication but is associated with a high perioperative mortality. Re do valve replacement surgery is associated with a mortality ranging from 37.5% to 54.5% for emergency cases and 8% to 20.3% for urgent cases[3]. Recently thrombolytic therapy has been successfully used to treat prosthetic valve thrombosis [4-6] and has emerged now as an alternative to re do surgery [7], [8] but dosage and duration of therapy have varied widely[4-7]. In this fluoroscopy and echo cardiography guided series, we studied the efficacy and safety of streptokinase therapy for patients with prosthetic valve thrombosis.

Material and Methods:

Patients: Elevan consecutive patients (4 men and 7 women) aged 14 to 52 years (mean 32.5±12.9 years) admitted in our institute on 12 occasions between January 1992 and October 1993 with prosthetic valve thrombosis formed the case material for

this study. All patients had clinical, echocardiographic and fluoroscopic evidence of valve thrombosis and did not have any known contraindication to thrombolytic therapy. One patient with prosthetic tricuspid valve had valve thrombotic episode twice at an interval of 8 months. The clinical criteria for prosthetic valve obstruction were taken as a recent onset of dyspnea or features of pulmonary venous hypertension in left sided valves and systemic venous hypertension in right sided valves associated with diminution or absence of prosthetic valve clicks with or without audible stenotic or regurgitant murmurs accross the prosthetic valve [9]. The cinefluoroscopic criteria of prosthetic valve dysfunction was taken as an opening angle less than 40 degrees, incomplete closure or no movement of disc [10]. Echo cardiographic features of prosthetic mitral valve obstruction was taken as a calculated mitral valve area less than $1.6cm^2$ and of aortic valve obstruction as a peak systolic gradient more than 50 mm Hg [11]. Prosthetic tricuspid valve obstruction was arbitrarily defined as a calculated valve area less than 1.6 cm^2. Of these 11 patients, 5 had prosthetic valve at mitral position, 2 had prosthetic valve at aortic position and 4 had prosthetic valve at tricuspid position. The type of valves were Bjork Shiley prosthesis in 7, Medtronic Hall valve in 3 and St. Jude Valve in one patient. The clinical details of all 11 patients are shown in table - 1.

Thrombosis developed 5.0±4.2 years after valve replacement surgery (range 3 months to 11 years). One patient with prosthetic aortic valve had undergone surgical thrombectomy earlier. While all patients with tricuspid valve thrombosis were in New York Heart Association (NYHA) Class II, 6 of 7 patients with left sided valve thrombosis were in class III and one patient with thrombosed aortic valve was in class IV. All patients had diminised/absent valve sounds. While 6 patients were in sinus rhytum, 5 were in atrial fibrillation.

Protocol: All patients underwent basal routine investigations which included hemogram, prothrombin time, electrocardiogram, chest rontgenogram, in addition to cinefluoroscopy and Doppler echocardiography. While valve opening angle was measured on fluoroscopy, valve area and mean and peak gradients across the obstructed valve were recorded on Doppler echocardiography.

Thrombolytic therapy was administered as intravenous streptokinase with a loading dose of 2.5 lakh units over 15 min. in 4 patients and 1 lakh units in 8 patients. This was preceeded by injection hydrocortisone 100mg intravenously. Maintenance infusion varied between 1 lakh U/hr/in 2 pts to 1000 U/Kg/hr in 9 patients. Duration of streptokinase was fixed as until successful thrombolysis is achieved or a maximum of 48 hr. Echocardiographic and fluoroscopic evaluations were done every 6 h. After completion of streptokinase therapy patients were started on Heparin 5000 U IV every 6 h and replaced by oral anticoagulants

over the next 2 to 5 days.

Results:

On admission to hospital 5 patients had an inadequate basal prothrombin time. Cinefluoroscopy had shown restricted disc motion in all patients. While all patients had significant gradients accross the obstructed valve, 5 patients (1 aortic and 4 tricuspid) had mild regurgitation accross the valve. Details of thrombolytic therapy are summarised in table - 2 and fluoroscopic and echocardiographic details in Table - 3. Mean duration of streptokinase therapy was 19.5±11.1 h (Range: 3 h to 38 h). Mean dose of streptokinase used was 13.0±7.1 L U (Range 3 lakh U to 26 Lakh U). Successful thrombolysis, defined as return of native sounds, disappearance of murmur, normalisation of fluoroscopic opening and closing angles and near normalisation of transvalvar gradients occurred in 11/12 cases (92%). One unsuccessful case was that of prosthetic tricuspid valve who had symptoms for four months and in him streptokinase infusion had to be stopped after 24 h due to gum bleeding. He underwent bidirectional Glen Shunt and is doing well now. One patient had rethrombosis after an interval of 8 months and was again successfully treated with streptokinase. There were no reactions associated with reuse of streptokinase.

Complications:-

One patients (case No.2) with prosthetic aortic valve developed a transient ischemic attack (TIA) (Right

hemiparesis), but recovered fully. Another patient (case No.6) developed a right hemiplegia after successful thrombolysis. While initial CT scan showed a pale infarct subsequently she developed an intracerebral hematoma needing neuro surgical intervention. She lapsed into Grade IV coma and hypotension and expired. There were no episodes of peripheral embolism. Minor bleeding manifestations were seen in 2/12 cases (17%). None required blood transfusion.

Follow - up: Follow up periodsvaried from 3 months to 18 months. But for the patient who developed rethrombosis at an interval of 8 months all other patients were doing well.

Discussion:

Thrombolytic therapy as first line of treatment for thrombosed prosthetic valves was attempted in this study. 11 of 12 events (92%) responded to thrombolytic therapy. This is in agreement with most reports,[5-7], but below the 100% success reported in one recent series of 16 cases [12]. Interestingly at surgery incidence of thrombosis as main cause of obstruction was 70% as described by Devari et al [13]. They suggested that failure or partial success of thrombolytic therapy may be a result of pannus formation which was seen in 11% of obstructed valves at surgery. Pannus was seen in association with thrombus in another 12% of cases [13].

Higher proportion of tricuspid valve cases in our series is because of endemicity of endomyocardial fibrosis

around our centre for which tricusped valve replacement is commonly performed. Generally, they presented with longer duration of symptoms of valve thrombosis and were in better functional class as compared to left sided valve thrombosis who present more acutely.

Dosage and duration of streptokinase therapy is yet to be standardised. While most studies used a bolus dose of 2.5 lakh U and a maintenance dose of 1 lakh units / hr, we found a bolus dose of 1 lakh U IV and a maintenance of 1000 U/Kg/h quite effective. Also duration of streptokinase infusion was shorter, 19.5±11.1 h (Range 3 - 38 h) in our series as compared to others, which ranged from 10 h to 112 h[5]. In one patient of rethrombosis, streptokinase was found to be effective after an interval of 8 months. This is not surprising as after 8 months anti streptokinase neutralising antibodies have been demonstrated to start declining[14].

Stroke either major or a transient ischemic attack has been documented to complicate thrombolytic therapy for thrombosed left sided prosthetic valves. In a review of 58 cases the incidence of embolic manifestations was 18%[6] while in another report the frequency was 20%[4]. We encountered it in 2 out of 7 (28%) cases of left sided valve thrombolysis. One was transient but the other patient developed a pale infarct which became hemorrhagic later, sufficient to produce death. While death despite thrombolysis is not uncommon, death due to stroke complicating thrombolytic therapy has been reported only once[6]. Minor bleeding

manifestation occurred in 2 patients (17%) which is similar to that reported in literature.

In conclusion this study demonstrates the efficacy of streptokinase therapy in prosthetic valve thrombosis but also highlights the possible fatal embolic / hemorrhagic episodes which can complicate the therapy of left sided prosthetic valves.

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Table - 1: CLINICAL CHARACTERISTICS OF PATIENTS PRESENTING WITH PROSTHETIC VALVE THROMBOSIS:

Case No.	Age	Sex	Diagnosis	Valve position	Valve model	Duration after surgery	Duration of Symptoms	NYHA Class	RHYTHM
1	16	F	RVEMF	Tricuspid	No.25 BSP	3 Months	1 Month	II	AF
2	44	M	AVD	Aortic	No.25 BSP	8 Years	7 days	IV	SR
3	41	F	RHD	Mitral	No.27 St.Jude	6 Months	7 days	III	AF
4	43	M	RVEMF	Tricuspid	No.25 Med-tronic	1 Year	4 months	II	AF
* 5	18	F	Ebstein's anomaly	Tricuspid	No.25 Med-tronic	1 Year	1 Month	II	SR
6	52	F	RHD	Mitral	No.25 BSP	7 Years	14 days	III	SR
7	39	M	Subaortic membrane	Aortic	No.23 BSP	11 Years	3 days	III	SR
8	39	F	RHD	Mitral	No.27 BSP	9 years	1 day	III	SR
* 9(5)	19	F	Ebstein's anomaly	Tricuspid	No.25 BSP	2 years	1 Month	II	SR
10	37	F	LVEMF	Mitral	No.25 BSP	7 Years	14 days	III	SR
11	14	F	RVEMF	Tricuspid	No.25 Medtronic	2 Years	1½ Months	II	AF
12	28	M	RHD	Mitral	No.29 BSP	11 years	14 days	III	AF

* 5 and 9 represent 2 thrombotic episodes in same patient

Abbreviations used: RVEMF=Right Ventricular Endo Myocardial Fibrosis, LVEMF= Left Ventricular Endomyocardial Fibrosis; AVD=Aortic Valve disease; RHD=Rheumatic heart disease; AF=Atrial Fibrillation, SR=Sinus Rhythm; BSP=Bjork Shiley Prosthesis.

Table - 2: Details of Thrombolytic Therapy Streptokinase

Case No.	Loading dose	Maintenance infusion	Duration of therapy	Total dose	Return of valve sounds	Outcome	Complications
1	1 Lakh U	1000 U/Kg/hr	30 h	19 Lakh U	24 h	Successful	...
2	2.5 Lakh U	ILU/hr	15 h	18 Lakh U	14 h	"	TIA
3	1 Lakh U	1000 U/Kg/hr	22 h	12 Lakh U	20 h	"	...
4	2.5 Lakh U	ILU/hr	24 h	26 Lakh U	..	Failure	Gum-bleeding
5	1 Lakh U	1000 U/Kg/hr	21 h	10 Lakh U	20 h	Successful	...
6	2.5 Lakh U	"	38 h	21 Lakh U	36 h	Expired after successful therapy	Hemorrhagic stroke
7	"	"	16 h	14 Lakh U	15 h	Successful	Gum-bleeding
8	1 Lakh U	"	3 h	3 Lakh U	2 h	"	...
9	1 Lakh U	"	13 h	8 Lakh U	13 h	"	...
10	1 Lakh U	"	7 h	5 Lakh U	4 h	"	...
11	1 Lakh U	1000 U/Kg/hr	36 h	15 Lakh U	36 h	"	...
12	1 Lakh U	1000 U/Kg/hr	9 h	5 Lakh U	8 h	"	...

Abbreviations used: TIA: Transient Ischemic attack

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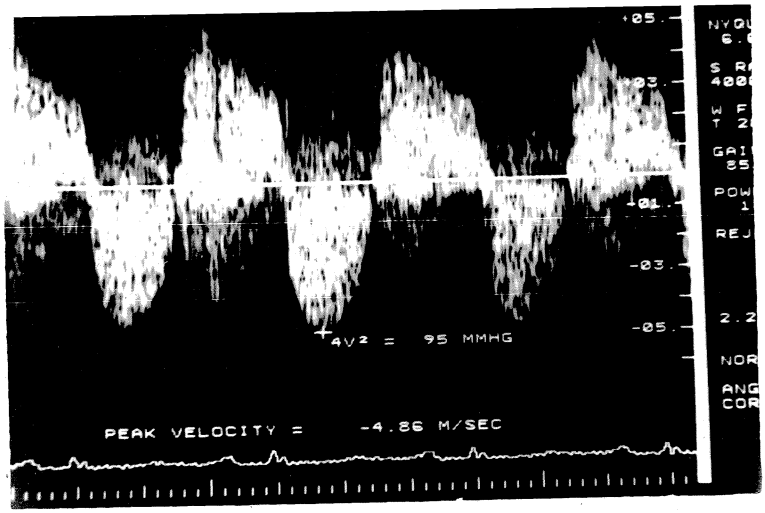

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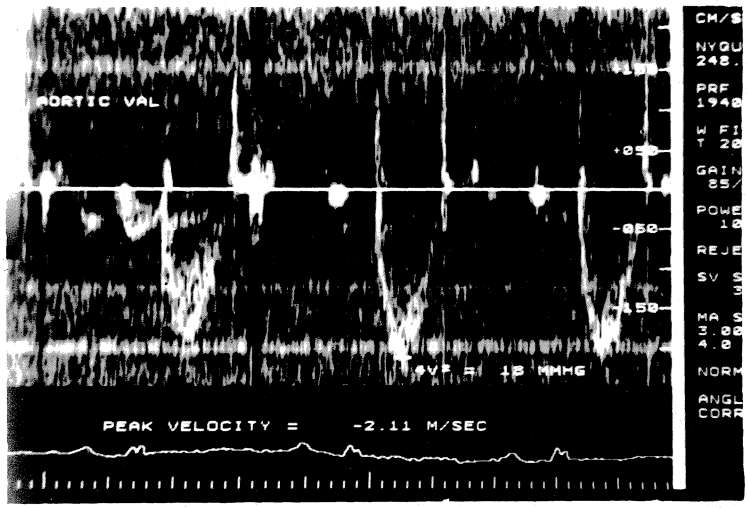

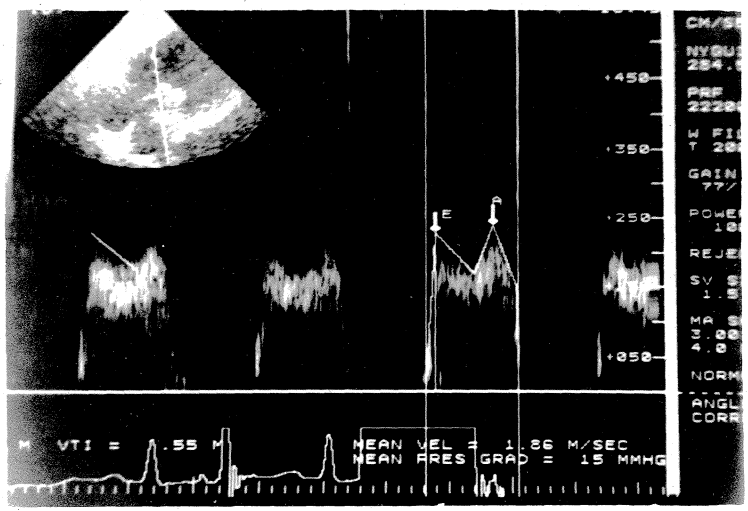

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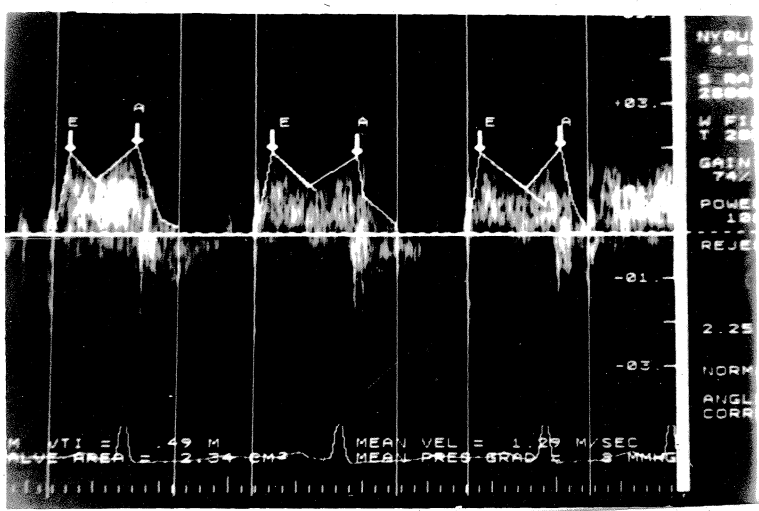
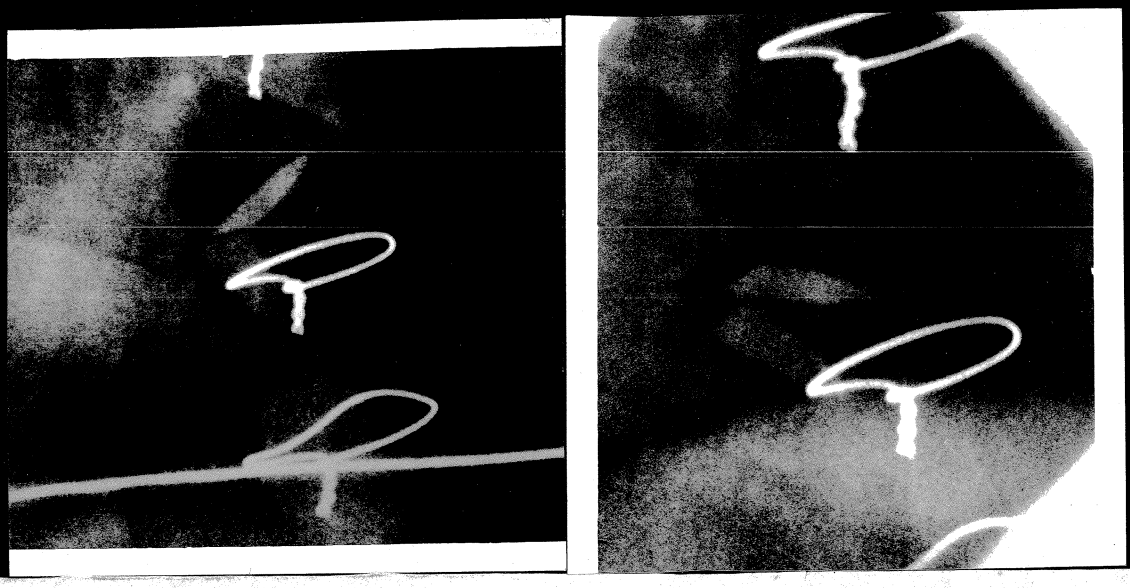
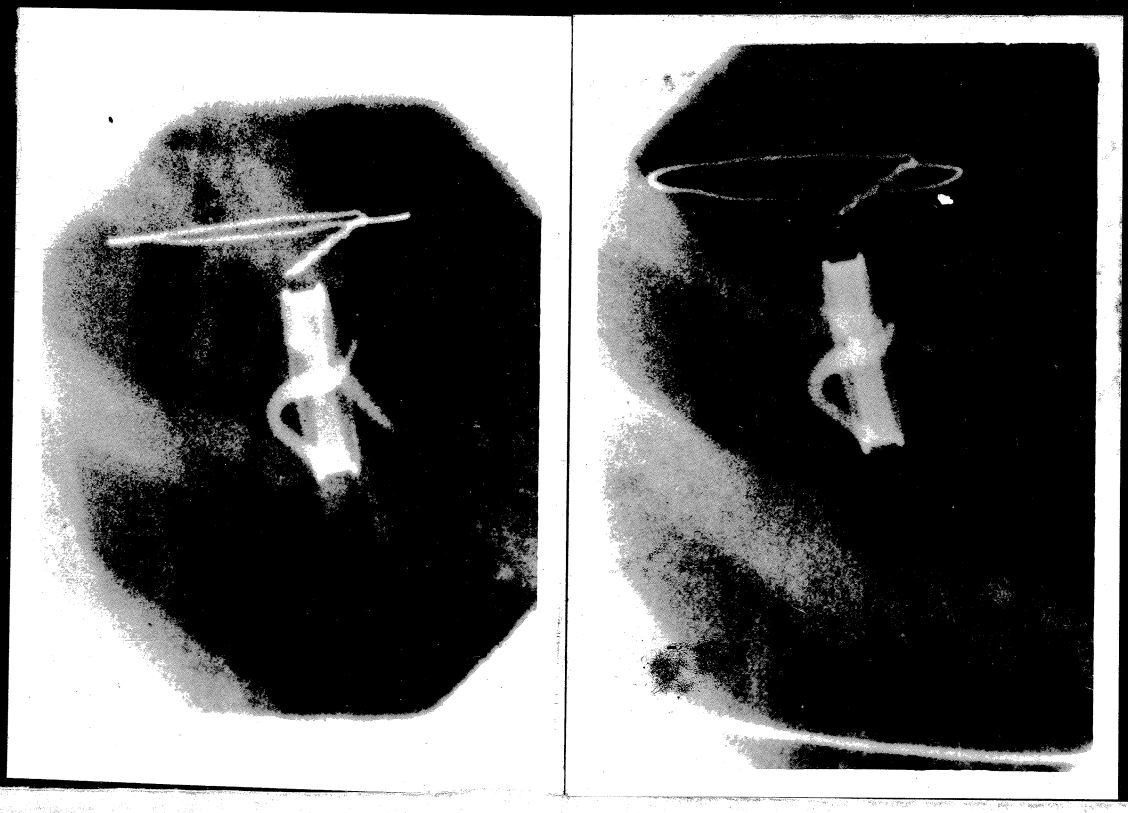




CINE FLUOROSCOPIC DIASTOLIC FRAMES OF St JUDE VALVE IN MITRAL POSITION BEFORE AND AFTER THROMBOLYTIC THERAPY. LATERAL LEAFLET WAS STUCK BUT OPENS FULLY AFTER SUCCESSFUL THROMBOLYSIS



CINE FLUOROCOPIC DIASTOLIC FRAMES OF MEDTRONIC HALL VALVE IN TRICUSPID POSITION BEFORE AND AFTER SUCCESSFUL THROMBOLYTIC THERAPY

~~LIST OF PROCEDURES DONE~~

PROJECT REPORT

TITLE OF THE PROJECT: PRIMARY PULMONARY HYPERTENSION: NATURAL HISTORY AND PROGNOSTIC FACTORS.

NAME D.RADJASSEGAR

PROGRAMME : D.M. (CARDIOLOGY)

MONTH & YEAR OF SUBMISSION : DECEMBER 1993.

CONTENTS

PRIMARY PULMONARY HYPERTENSION: NATURAL HISTORY AND PROGNOSTIC FACTORS

SUMMARY

A retrospective cohort study was carried out in 61 patients (30 males, 31 females, age 24.6 ± 11.8 years) with primary pulmonary hypertension diagnosed by strict clinical and hemodynamic criteria, to obtain an understanding of the natural history and prognostic markers. While 15 patients were alive, 46 patients (76%) had expired during the follow-up period. Two, five and ten year survivals were 48%, 32% and 12% respectively. Median survival duration from time of diagnosis was 22 months. The survivors had significantly higher age of onset, cardiac index and significantly lower right atrial mean pressure, right ventricular end diastolic pressure, cardio thoracic ratio from chest rontgenogram and calculated pulmonary vascular resistance as compared to non survivors. While pulmonary artery systolic pressure was not significantly different, pulmonary artery diastolic and pulmonary artery mean pressures were significantly lower in survivors than in non-survivors. Lower New York Heart Association class, right atrial mean pressure ≤ 7mm Hg, right ventricular end diastolic pressure ≤ 10 mm Hg, cardiac index > 2.5 $L/min/m^2$, pulmonary arterial oxygen saturation > 60%, were associated with significantly longer survival. The degree of pulmonary arterial hypertension had an indirect prognostic effect through the above parameters. Vasodilator therapy did not significantly alter the outcome of patients with primary pulmonary hypertension.

Key Words: Primary Pulmonary Hypertension, Hemodynamics, Natural history, Prognosis.

INTRODUCTION

Primary pulmonary hypertension (PPH) is widely accepted to be a progressive, incurable and fatal illness. Most of the natural history studies have found the average survival to be less than five years.[1-4] Data from the National Institute of Health (NIH) National registry for PPH is now available.[5,6,7] However reports on patients with primary pulmonary hypertension who have survived 10 years and longer are also available in the literature.[8,9] Large series have been reported from India which deal with clinical [10,11,12] or autopsy data[13] on PPH. In this study we retrospectively analysed the natural history and clinical and hemodynamic prognostic markers of patients with PPH in our centre.

MATERIAL AND METHODS

Between 1977 and 1991, 84 patients were diagnosed to have PPH by cardiac catheterisation at our centre. (35 males, 49 females, mean age 24.8 ± 11.8 years, range 3-55 years). The clinical and hemodynamic features of all these patients confirmed to the diagnostic criteria for PPH proposed by Kanemoto in 1979.[4] Minimum of 2 year follow up was required to be included in the study. Of the 84 patients, 23 were excluded because complete follow-up information was not available. Thus, 61 patients (30 males, 31 females, mean age 24.6 ± 11.8 years, range 3-53 years) constituted our study group.

Clinical Features:

Clinical records of all patients were reviewed. Noted particularly were age at onset of symptoms, age at the time of diagnosis, interval from initial clinical symptoms to diagnosis, history of syncope, right heart failure (RHF), New York Heart Association (NYHA) functional class at time of diagnosis and evidence of tricuspid regurgitation(TR) and pulmonary regurgitation(PR) by auscultation. Cardiothoracic rato(CTR) by chest rontgenography and presence of right ventricular hypertrophy(RVH) by electro cardiogram were also noted. Details about vasodilator therapy were also noted.

Hemodynamic data

During cardiac catheterisation, systemic arterial oxygen saturation, pulmonary arterial oxygen saturation, right atrial mean pressure (RAMP), right ventricular systolic (RVSP) and end diastolic pressures (RVEDP), pulmonary artery systolic, diastolic and mean pressures (PASP, PADP, PAMP), pulmonary artery wedge pressure (PAWP) and systemic arterial pressure were measured. Presence of patent foramen ovale (PFO) was also noted. From these data standard formulae were used to calculate cardiac index (CI), pulmonary vascular resistance index (PVRI), total pulmonary resistance (TPR) and right ventricular work index (RVWI). RVWI was calculated by using the formula,

$$RVWI = \text{Pulmonary Index} \times (PAMP - RVEDP) \times 0.0136 kg/min/m^2.$$

Also derived were ratios of pulmonary vascular resistance to systemic vascular resistance and pulmonary artery systolic pressure to systemic systolic pressure.

Follow-up

All patients were followed-up for a minimum period of 2 years or until death. Follow-up information was obtained by repeat clinical examinations at our clinic or from responses to letters to the patients or relatives and to the referring physicians.

Statistical analysis

For group comparisons, student 't' test was used for continuous or interval variables and Chisquare test for categorical variables.

Survival analysis using life table method was done and the survival curve plotted. Univariate comparison of survival was done using the Lee Desu statistic.

Pearson's correlation coefficient 'r' was computed for survival months and various clinical and hemodynamic variables.

All statistical procedures were done using SPSS/PC + version 4.0.

RESULTS

Clinical features

The patient group consisted of 30 males (49%) and 31 females (51%). At entry into the study, the mean age was 24.6 years. Mean duration of symptoms was 30 months, Sixty-two percent of patients were in the second or third decades of life. At the time of diagnosis, the clinical features were dyspnea 94%, fatigue 75%, syncope 29.5%, dizziness 50%, hoarseness of voice 10%, hemoptysis 6% and Raynaud´s phenomenon 5%. History of right heart failure (RHF) was present in 26%. Eight percent were in NYHA class I, 49% in NYHA II, 40% in NYHA III and 3% in NYHA IV. Among physical findings, 44% had TR and 26% had PR by ausculation. While mean CTR was 0.58 ± 0.06, RVH by ECG was seen in 88%. Sixty one percent of the patients received vasodilator therapy. The clinical features are summarised in Table-1.

Hemodynamic Features (Table - 3)

Pulmonary arterial hypertension (PAH) was mild in 2, (pulmonary artery mean pressure between 20-30 mm Hg) moderate in 17 (PAMP between 31-49 mm Hg) and severe in 42 patients (PAMP 50 mm Hg or above). PA pressure was suprasystemic in 24 patients 39%). Aortic arterial oxygen desaturation (< 95%) was seen in 32 patients (Mean 93.3 ± 3.7% ; Range 80 to 99%). Pulmonary arterial oxygen saturation less than 60% was seen in 24 patients. (Mean 63.4 ± 11.1 Range 30 to 83%). Cardiac index was less than 2.5 L/min/m^2 in 34 patients (56%). Satisfactory pulmonary wedge

tracing was obtained in 75% of patients and in 15% of patients left atrium was entered via a patent foramen ovale.

Right heart filling pressures were normal (RA mean <7 mm Hg) in 32 patients (52%). Calculated, total pulmonary resistance (TPR) was > 15 wood um^2 in 43 patients (70.5%).

Clinical Course

At the time of last follow-up information, 46 patients (75.4%) had died (Fig.1). Among the 15 surviving patients 2 had mild, 4 had moderate and 9 had severe PAH at the time of initial hemodynamic study. While survival was 100% in mild PAH, only 24% of patient with moderate PAH and 22% of patients with severe PAH were alive. Two of the surviving patients had suprasystemic PAH and were alive 2 years and 10 years after hemodynamic study. The longest surviving patient was alive 21 years from onset of symptoms and 11 years after hemodynamic diagnosis. This patient had a pulmonary artery mean pressure of 50 mm Hg and PVRI of 22.5 wood units at hemodynamic study. The median interval from diagnosis to death was 13 months (Range 1-126 months), and 70% of deaths occured within 2 years after diagnosis. The contributing factors, causes or modes of death were RHF in 57% and sudden death in 41%. In one patient death was related to cardiac catheterisation (2.2%).

Prognostic factors:

A number of clinical and hemodynamic variables were tested for prognostic importance by univariate analysis (Table 3). Best

prognostic factors were CTR, PA saturation, RAMP, RVEDP, PA/AO systolic pressure ratio, TPR, PVRI and PVR/SVR ratio. While absolute level of RVSP and PASP did not differentiate between survivors and non survivors, PADP and PAMP were significantly lower in the survivors as compared to non-survivors meaning thereby that survivors had a wider pulmonary arterial pulse pressure. By Chisquare analysis PR,TR, syncope and vasodilator therapy did not differ significantly between survivors and non survivors. Survial analysis using life table method for various variables were done (Univariate comparison) using Lee Desu statistic. The results are summarised in table 4.

Clinical and hemodynamic data in patients who died within 2 years of catheterisation were compared with those who died after 2 years (Table 5). While absolute PA pressures were not significantly different, cardiac index, PA oxygen saturation, right heart filling pressures and calculated PVRI and TPR were significantly different in the two groups.

Similar comparison was done between patients who survived less than 12 months and those who survived 13-24 months from hemodynamic study. While CTR was significantly higher, CI, aortic saturation, PA saturation and calculated RVWI were significantly lower in patients who died early (Table 6).

Hemodynamic parameters in patients who died suddenly and those who died of right heart failure were compared. While CI and PA oxygen saturation were significantly higher in patients

who died suddenly, RAMP and RVEDP were significantly higher in patients who died of right heart failure. Other parameters did not differ significantly (Table 7).

Finally, the relationship between hemodynamic parameters and the number of post catheterisation survival months was studied. While cardiac Index ($r = 0.467$; $p < p.001$), aortic saturation ($r = 0.390$; $p < 0.001$) and PA saturation ($r = 0.536$; $p < 0.001$) correlated positively, CTR ($r = 0.453$; $p < 0.001$), RAMP ($r = 0.345$; $p < 0.01$), RVEDP ($r = 0.374$; $p < 0.01$), PVRI ($r = 0.403$; $p < 0.001$) and TPR ($r = 0.409$; $p < 0.001$) correlated inversely with survival months. RVSP, PASP, PADP and PAMP did not correlate with survival duration.

DISCUSSION

This clinical series of 84 patients with PPH diagnosed on the basis of rigid clinical and hemodynamic criteria out of 12,000 cardiac catheterisations done in our institute during this period forms 0.7% of all hemodynamic studies which is much less than the 1.5% reported from Vellore.[10] This may be due to the fact that, of late, the patients with severe PAH, with no or mild cyanosis are being investigated with two dimensional and doppler echocardiography, contrast echocardiography and blood gas studies to defer invasive procedure. Mean age at presentation, 22.3 ± 11.7 years is similar to that reported in literature.[1] Eighty percent of patients were in second, third or fourth decades of life. Youngest patient was 3 years old at presentation and 8%

were in first decade of life. In our series 58% were females and 42% were males. This is different from that reported in literature where 70-80% of patients are females.[1-4] Higher male incidence and male preponderence in tropics has however been reported.[11] Interestingly, follow-up data was available in 86% of males (30/35) while only 63% (31/49) of females came for follow-up. The frequency of various clinical features were in agreement with that available in literature.[1] Only 61% of patients received vasodilator therapy as it was not routinely prescribed to our patients in early years. That majority of our patients had advanced form of disease is evident from the fact that PAH was severe (PAMP > 50 mm Hg) in 67% and suprasystemic in 39%. Only 2 patients (3.3%) had mild PAH. Even in terms of TPR, 70.5% of our patients had TPR > 15 wood Um^2. Catheterisation related mortality of 2.2% is lower than the 5% reported by others.[1] As many as 46 patients (76%) died during follow up, which is in agreement with the natural history of this disease.

Mean survival of 28.1 months from the time of diagnosis and 53.6 months from the onset of symptoms, is short and is in agreement with that reported by others.[1-4] While 33% died within 1 year and 52% within 2 years of diagnosis, 8 patients (16%) and 6 patients (12%) had lived for more than 10 years from onset of symptoms and diagnosis respectively. Longest survival was 130 months from diagnosis and 250 months from onset of symptoms. Such instances are however reported.[9] There was a higher incidence of sudden deaths in our series, 41%. This is much

higher than 7% reported in Mayoclinic series[1] but in agreement with the 37% reported in a Japanese survey.[4] However no parameter could significantly identify patients at risk of sudden death. As has been the experience with others[1,2] cardiac index, right heart filling pressures (RAMP, RVEDP) and calculated PVR (TPR, PVRI) were better prognostic markers to differentiate suvivors from non survivors than the absolute level of PAH. Though PASP was not different in survivors and non survivors, PADP and PAMP were significantly lower in survivors meaning thereby that wider pulmonary artery pulse pressure is prognostically better. Our patients had severe degree of PAH and pulmonary vascular disease and would fall into the category of non responders to vasodilators as predicted by Lupi Herrara[14] in his study where responders to hydralazine had only mild to moderate degree of PAH. Hence as expected, vasodilator therapy did not significantly alter the natural history of the disease. Among patients who died, longer survival (> 24 months) was identified by higher cardiac index, lower right heart filling pressures and lower calculated PVR rather than the severity of PAH (Table 5). Likewise the same factors correlated with the number of survival months. By life table analysis, RA mean pressure $\leq$ 7 mmHg, RVEDP $\leq$ 10 mmHg, CI > 2.5 L/min/m^2, PA saturation > 60%, aortic saturation > 95% identified significantly longer survival. Interestingly, age at onset and diagnosis was significantly lower in non survivors as compared to survivors ($p = 0.02$), thereby meaning a poorer prognosis with earlier onset. However most studies did not attach prognostic significance to age of onset.

In conclusion, our study demonstrates that, prognosis in PPH is largely determined by the integrity of right ventricle as evidenced by a higher CI, lower right heart filling pressure, higher PA oxygen saturation and lower calculated PVRI & TPR. Though most patients die early, long survival (> 10 years) is possible.

Non availability of autopsy information is however a short coming of this study.

ACKNOWLEDGEMENTS

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TABLE 1

BASELINE CLINICAL CHARACTERISTICS OF PATIENTS PRESENTING WITH PPH

	All patients n = 84	Patients in whom current status is known n = 61
1. Age at onset (years)	22.5 ± 11.7	22.3 ± 11.7
2. Age at Diagnosis (years)	24.8 ± 11.8	24.6 ± 11.8
3. Duration of symptoms (months)	27.4 ± 24.5	30.0 ± 27.6
4. h/o syncope	21 (25%)	18 (29.5%)
5. h/o Right heart failure	20 (24%)	16 (26.2%)
6. RVH by ECG	76 (90%)	54 (88%)
7. CTR by CXR	0.58 ± 0.06	0.58 ± 0.06
8. Vsodilator therapy	48 (66%)	37 (61%)

RVH = Right ventricular hypertrophy;

CTR = Cardio thoracic ratio on chest rontgenogram.

TABLE 2

BASELINE HEMODYNAMIC CHARACTERISTICS OF PATIENTS PRESENTING WITH PPH

Variable	All patients n = 84 Mean ± SD	Patients in whom current status is known n = 61 Mean ± SD
1. CI L/min/m^2	2.63 ± 1.14	2.52 ± 0.83
2. Aortic oxygen saturation (%)	93.2 ± 3.4	93.3 ± 3.7
3. PA oxygen saturation (%)	62.8 ± 10.7	63.4 ± 11.1
4. RAMP (mm Hg)	9.0 ± 5.2	8.7 ± 5.3
5. RVEDP (mm Hg)	13.0 ± 6.4	12.5 ± 6.6
6. PASP (mm Hg)	101.1 ± 27.8	99.3 ± 27.4
7. PAMP (mm Hg)	65.2 ± 19.2	63.7 ± 18.0
8. PAWP (mm Hg)	7.9 ± 1.8	7.7 ± 1.8
9. PVRI (Wood units)	26.0 ± 14.8	25.4 ± 13.1
10. TPR (Wood units m^2)	23.8 ± 14.2	22.7 ± 13.8
11. PVR/SVR Ratio	0.75 ± 0.27	0.74 ± 0.26
12. RVWI Kg/min/m^2	1.72 ± 0.80	1.73 ± 0.85

CI = Cardiac Index; RAMP = Right atrial mean pressure;
RVEDP = Right ventricular end diastolic pressure;
PASP, PAMP & PADP = Pulmonary artery systolic, mean and diastolic pressures;
PAWP = Pulmonary artery wedge pressure;
PVRI = Pulmonary vascular resistance index;
TPR = Total pulmonary resistance;
PVR/SVR = Pulmonary vascular resistance to systemic vascular resistance ratio;
RVWI = Right ventricular work Index.

TABLE 3

UNIVARIATE ANALYSIS OF PROGNOSTIC FACTORS

	Survivors (n = 15)	Non-Survivors (n = 46)	P value
1. Age at onset (yrs)	27.7 ± 13.1	20.5 ± 10.7	0.035
2. Age at diagnosis (yrs)	30.7 ± 12.6	22.6 ± 10.9	0.019
3. CTR	0.54 ± 0.04	0.59 ± 0.06	0.003
4. RVH (ECG)	Present in 73%	Present in 96%	0.045
5. CI ($L/min/m^2$)	2.91 ± 0.89	2.39 ± 0.79	0.038
6. Aortic saturation (%)	95.0 ± 2.5	92.8 ± 3.9	0.046
7. PA saturation (%)	70.8 ± 8.7	61.0 ± 10.8	0.002
8. RA mean pressure(mm Hg)	6.0 ± 3.3	9.5 ± 5.6	0.023
9. RVEDP (mm Hg)	8.7 ± 4.2	13.8 ± 6.8	0.009
10. PASP (mm Hg)	87.7 ± 23.0	103.0 ± 27.9	P=NS
11. PADP (mm Hg)	40.0 ± 10.9	50.6 ± 14.3	0.01
12. PAMP (mm Hg)	54.9 ± 16.7	66.6 ± 17.7	0.028
13. PA/AO systolic pressure	0.73 ± 0.2	0.94 ± 0.24	0.004
14. PVRI (Wood U)	18.2 ± 9.9	27.7 ± 13.3	0.013
15. TPR (Wood U. m^2)	14.4 ± 6.5	25.4 ± 14.5	0.006
16. PVR/SVR Ratio	0.57 ± 0.2	0.80 ± 0.26	0.003

Abbreviations are the same as in Tables 1 & 2.

TABLE 4

LIFE TABLE ANALYSIS OF SURVIVAL DURATION (MONTHS)

COMPARISON OF SURVIVAL USING THE LEE DESU STATISTIC

Variable	No.	Median Survival (months)	Survival (%) 2year	5year	10year	P Value
1. Vasodilator therapy						
+	37	21	46%	34%	21%	P = NS
-	24	30	50%	24%	0%	
2. H/o Right Heart failure						
+	16	8	6%	6%	6%	P<0.0001
-	45	53	62%	41%	15%	
3. NYHA Class						
I	5	126	100%	100%	67%	
II	30	35	62%	36%	12%	
III	24	11	15%	15%	5%	P=0.0001
IV	2	9	0%	0%	0%	
4. RAMP (mm Hg)						
≤ 7	32	63	73%	46%	22%	P=0.005
> 7	29	13	14%	14%	4%	
5. CI L/min/m^2						
≤ 2.5	34	29	27%	14%	7%	P=0.005
> 2.5	27	17	71%	52%	20%	
6. Aortic saturation						
< 95	32	27	41%	21%	7%	P=0.0045
≥ 95	29	80	45%	42%	17%	
7. PA saturation						
≤ 60	24	11	17%	9%	5%	P=0.0001
> 60	37	63	68%	45%	18%	
8. RVEDP (mm Hg)						
≤ 10	29	63	71%	43%	15%	P=0.0002
> 10	32	13	27%	20%	10%	

Abbreviations are the same in tables 1 & 2.

TABLE 5

CLINICAL AND HEMODYNAMIC DATA IN PATIENS WHO DIED WITHIN 2 YEARS COMPARED WITH THOSE WHO DIED MORE THAN 2 YEARS FROM THE TIME OF CATHETERISATION

	Variable	Survival ≤ 2 year n = 32	Survival > 2 year n = 14	P Value
1.	H/o Right heart failure	Present in 47%	0%	0.005
2.	CTR	0.60 ± 0.06	0.56 ± 0.05	0.014
3.	CI ($L/min/m^2$)	2.1 ± 0.60	3.05 ± 0.82	0.001
4.	PA saturation (%)	57.9 ± 9.8	68.1 ± 9.9	0.002
5.	RAMP (mm Hg)	11.2 ± 5.8	5.9 ± 2.2	0.002
6.	RVEDP (mm Hg)	15.7 ± 7.1	9.4 ± 3.2	0.003
7.	PVRI (Wood U)	33.3 ± 13.3	19.5 ± 9.3	0.004
8.	TPR (Wood U. m^2)	28.7 ± 15.0	17.7 ± 0.18	0.016
9.	RVWI ($Kg/min/m^2$)	1.53 ± 0.79	2.13 ± 0.78	0.023

Abbreviations are the same as in Tables 1 & 2.

TABLE 6

COMPARISON OF VARIABLES IN PATIENTS WHO SURVIVED < 12 MONTHS (n = 20) WITH THOSE WHO SURVIVED 13-24 MONTHS (n = 12) AFTER CATHETERISATION

	Variable	Survival ≤ 12 months n = 32	Survival > 12 months n = 14	P Value
1.	CTR	0.63 ± 0.05	0.56 ± 0.03	< 0.001
2.	CI L/min/m^2	1.98 ± 0.61	2.34 ± 0.48	< 0.001
3.	Aortic saturation (%)	91.0 ± 4.4	93.8 ± 2.7	< 0.05
4.	PA saturation	54.7 ± 9.8	63.2 ± 7.5	0.01
5.	RVWI (Kg/min/m^2)	1.26 ± 0.71	1.99 ± 0.71	< 0.01

Abbreviations are the same as in tables 1 &2.

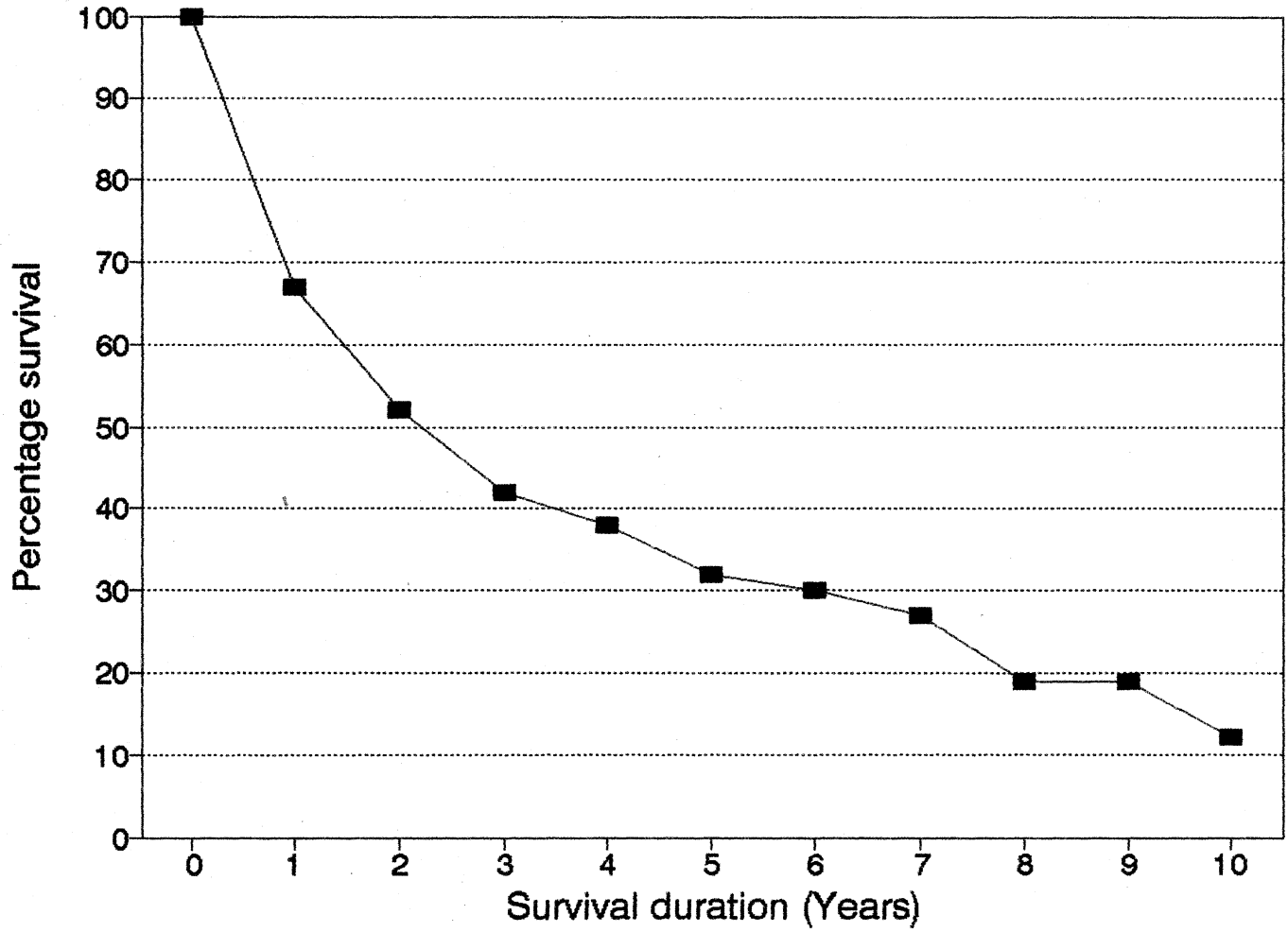
TABLE 7

HEMODYNAMIC PARAMETERS IN PATIENTS WHO DIED SUDDENLY (n=19) COMPARED WITH THOSE WHO DIED OF RIGHT HEART FAILURE (RHF) (n=26)

Variable	RHF n = 26	Sudden death n = 19	P Value
1. CI	2.17 ± 0.68	2.66 ± 0.84	0.034
2. PA Saturation (%)	57.3 ± 10.8	65.1 ± 8.5	0.012
3. RAMP (mm Hg)	11.2 ± 6.5	7.3 ± 2.9	0.018
4. RVEDP (mm Hg)	15.5 ± 8.1	11.4 ± 3.5	0.043
5. RVWI ($Kg/min/m^2$)	1.46 ± 0.67	1.99 ± 0.90	0.029

Abbreviations are the same as in tables 1 & 2.

Figure 1: Graph of survival function
from the time of hemodynamic study



~~LIST OF PROCEDURES DONE~~

PROJECT REPORT

TITLE OF THE PROJECT: IMMEDIATE AND FOLLOW-UP HEMODYNAMIC RESULTS OF PERCUTANEOUS BALLOON MITRAL VALVOTOMY.

NAME ..... D.RADJASSEGAR

PROGRAMME : D.M. (CARDIOLOGY)

MONTH & YEAR
OF SUBMISSION : DECEMBER 1993.

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY, TRIVANDRUM 695 011

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Date	

CONTENTS

Immediate and Follow up Hemodynamic Results of Percutaneons Balloon Mitral Valvotomy

SUMMARY

Thirty two patients (27 females and 5 males) age ranging from 10-47 years underwent Balloon mitral valvotomy (BMV) either with single balloon (23 patients) or with double balloon (9 patients). 24 patients were in New York Heart Association (NYHA) Class II and 8 were in NYHA Class III. The ratio of Effective Balloon Dilatation Area to Body surface area (EDBA/BSA) was 3.79±0.3. After dilatation, mitral valve area (Gorlin's formula) increased from 0.74±0.23 CM^2 to 1.8±0.63 CM^2 ($P < 0.001$). Mean diastolic gradient decreased from 15.8 ± 5.9 mm Hg to 4.2±2.9 mm Hg ($P < 0.001$). Pulmonary artery mean pressure decreased from 35.4±12.8 to 27.5 ± 10.4 mm Hg ($P < 0.001$. The procedure was well tolerated by all patients. 2/32 patients (6.3%) had poor initial result and were recomended closed surgical commissurotomy. 24 patients underwent repeat cardiac catheterisation at an interval of 11 ± 3.4 months after BMV. 22 were in NYHA class I and 2 were in NYHA Class II. Mitral valve area by Gorlin's formula showed a mild fall though not significant. (1.78 ± 0.58 to 1.53±0.44 cm^2 P = NS). There was a significant increase in cardiac index and mean transmitral gradient. Pulmonary artery systolic and mean pressures continued to fall significantly. Restenosis was seen in 2 patients (8.3%). BMV produces good immediate results which is maintained during short term follow up.

Introduction

Percutaneous balloon mitral valvotomy produces prompt improvement of symptoms for many patients with mitral stenosis. [1-6] Studies comparing BMV with closed surgical commissurotomy have shown comparable improvement in symptoms - and similar increase in mitral valve area calculated using Gorlin's formula[7]. However follow up results are needed to evaluate the long term usefulness of BMV. This study focuses on the follow up symptomatic status, and hemodynamic data at a mean interval of 11 months after BMV in 24 out of 32 patients who have had a repeat cardiac catheterisation. NYHA class and hemodynamic data are compared in the same patients, immediately before and after BMV.

Material and Methods:

The aim of this study was to collect follow up hemodynamic data of patients who underwent BMV in our institution in 1991 as close to 1 year after procedure as possible. In all 32 patients underwent BMV during the period. There were 27 females and 5 males. Their age ranged from 10 to 47 years. (Mean 27 + 10 years). Thirty one patients were in sinus rhythm and one was in atrial

fibrillation. 24 were in NYHA Class II and 8 were in NYHA Class III. None of the patients had calcification of mitral valve on fluoroscopy. Two patients underwent BMV for restenosis following a previous closed surgical commissurotomy. Pre BMV echocardiographic scoring of the mitral valve was done as described[8]. 31 patients had a score less than 8 and one patient had a score of 9. Two patients had associated mild aortic regurgitation. Five patients had mild mitral regurgitation pre BMV.

Patients were re catheterised at a mean period of 11 ± 3.4 months (Range 8-17 months) after BMV. 24 patients (5 males and 20 females, Mean age 26 ± 9 years) returned for repeat catheterisation and the remaining are planned for restudy in the near future. Since restudy was advised electively for all patients, the data collected is likely characteristic of the entire patient population.

Procedure:

Written informed consent was obtained from each patient prior to BMV. BMV was performed as previously described[2]. Effective Balloon dilatation area (EDBA) was determined by standard geometric analysis from balloon

diameter as described earlier[9]. Aim was to achieve a EDBA/BSA of 3.75 to 4.0. Mean EDBA/BSA was 3.79 ± 0.3. Atrial septal dilatation was done with 8 mm balloon in 18 patients and 10 mm balloon in 14 patients. Single balloon was used in 23 patients and double balloon in a 9 patients. Mean EDBA/BSA was 3.73 ± 0.26 with single balloon and 3.97 ± 0.29 with double balloon. The ratio was significantly higher with use of double balloon ($P < 0.05$). Mean transmitral diastolic gradient, pulmonary artery pressures, cardiac index, mitral valve area by Gorlin's formula, Left to Right atrial level shunt and angiographic mitral regurgitation were assessed before, after BMV and during follow up cardiac catheterisation.

Statistical Analysis:

Paired student's 't' test was applied to compare the immediate and follow up results of BMV. Unpaired 't' test was applied to compare the results of single balloon with double balloon technique.

Results:

Immediate results: BMV was performed in all the 32 patients in whom it was attempted. Immediate results were

good. There was significant fall in mean diastolic gradient, pulmonary artery systolic and mean pressures and significant increase in mitral valve area and cardiac index (Table-1). Mitral valve area increased from 0.74 ± 0.23 cm^2 to 1.8 ± 0.63 cm^2 ($p < 0.001$). Mean diastolic gradient decreased from 15.8 ± 5.9 to 4.2 ± 2.9 mm Hg ($p < 0.001$): Cardiac index increased from 2.61 ± 0.75 $L/min/m^2$ to 2.75 ± 0.65 $L/min/m^2$ ($p < 0.05$); Pulmonary artery systolic pressure decreased from 53 ± 18.8 to 42 ± 15.7 mm Hg ($p < 0.001$). As a group there was no significant difference in the outcome regarding mitral valve area and mean diastolic gradient, whether single balloon or double balloon was used (Table 2). Good result, defined as post dilatation mitral valve area more than 1.5 cm^2 or more than 100% increase in mitral valve area[10] was seen in 27 out of 32 cases. Poor result, defined as final mitral valve area less than 1 cm^2 or less than 25% increase in mitral valve area or final mean diastolic gradient more than 10 mm Hg was seen in 2 patients. In both of these patients single balloon technique was used. Remaining 3 patients had suboptimal result with final valve area less than 1.5 cm^2 or calculated increase in mitral valve area between 25 to 100%. Both the patients who had poor initial result of

BMV, underwent closed surgical commissurotomy. There was no mortality associated with the procedure. Mild MR was seen on post dilatation LV angiogram in 10 out of 32 patients. 5 of these 10 patients had mild MR on pre dilatation LV angiogram itself. There was no incidence of severe MR. None of the patients developed thrombo embolic events. 2 patients developed pericardial collection (hemopericardium) without tamponade. Both were managed conservatively. They did not need pericardiocentesis. 10/32 patients had calculable left to right shunt on immediate post BMV oximetry run and QP/QS ratio was 1.2:1 in 8 patients. 2 patients had shunt ratio of 1:5:1.

Follow up results:

Of the 24 restudied, prior to BMV 6 were in NYHA class III and 18 were in NYHA class II. (Fig.1) At follow up 2 were in NYHA class II and 22 were in NYHA class I. Table 3 shows the hemodynamic data at follow up in 24 patients compared with pre BMV and immediate post BMV values. There was significant increase in cardiac index during follow up. There was continuing fall in pulmonary artery systolic pressure and pulmonary artery mean pressure (Fig.2). There was a significant increase in

mean transmitral gradient. Calculated mitral valve area by Gorlin's formula decreased from 1.78 ± 0.58 cm^2 to 1.53 ± 0.44 cm^2 (Fig.3). Restenosis, defined as loss of more than 50% of the initial gain in mitral valve area[10] occured in 2/24 patients(8.3%). Angiographic mild MR was seen in 4 patients and calculable Left to Right atrial level shunt of 1.2:1 was seen in 8 patients. No patient had shunt ratio more than 1.5:1.

Discussion:

Our study shows that BMV when performed in selected patients of mitral stenosis with low echo score produces good immediate results. The procedure was completed in all the patients without any mortality or major complications. 27/32 patients (85%) achieved good immediate result. This is similar to 88% reported by Palacious et al[10] and 73% reported by Desideri et al[11]. 31 out of 32 patients in this series had Echo score of mitral valve less than 8 meaning good selection of cases. The procedure produced poor result in 2 patients (6.3%) and in the remaining 3 the results were sub-optimal. Mild angiographic MR was seen in 10/32 patients (31.2%) which is similar to that reported in literature[10, 11].

Calculable left to Right atrial level shunt was seen in 10 out of 32 although in all QP/QS ratio was about 1.2:1.

Few follow up hemodynamic[10,12] and echocardiographic studies[6,11,13,14] after BMV are available in literature (Table-4). In our study, 23 out of 24 patients (96%) showed symptomatic improvement of at least one NYHA class at 11 months follow up. Only 2 out of 24 patients (8.3%) restudied had evidence of restenosis, with the reported figure in literature ranging from 0% to 21%. Block et al[12] reported that immediately after BMV there is overestimation of mitral valve area by Gorlin's formula due to decompression of LA via the atrial septal defect. However during follow up most ASDs close leading to a mild decrease in mitral valve area. So restenosis calculated by hemodynamic study was higher (51%) than by echo study (16%). Our study also shows a fall in mitral valve area during follow up though not statistically significant. There was a significant increase in mean diastolic gradient on follow up study. This may be partly due to the fall in mitral valve area and partly due to the increased cardiac index. During follow up left ventricular angiogram, MR disappeared in 4 patients. The possible mechanism for decrease in MR are:

(1) reversible mitral valve stretching by BMV (2) fibrosis and healing of the end of commissures which may diminish MR (3) Improvement in the transient papillary muscle dysfunction produced by balloon trauma at the time of BMV[10].

In conclusion, BMV produces excellent immediate results with low complication rate. The results are maintained at short term follow-up with low incidence rate of restenosis.

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Table-1

Immediate Hemodynamic Results of BMV in 32 Patients

	Pre dilatation	Post dilatation	P-Valve
1. Mitral valve area(cm2)	0.74±0.23	1.8±0.63 cm2	<0.001
2. Mean diastolic gradient(mm Hg)	15.8±5.9	4.2±2.9	<0.001
3. Pulmonary artery systolic pressure (mm Hg)	53.1±18.8	42±15.7	<0.001
4. Pulmonary artey mean pressure (mm Hg)	35.4±12.8	27.5±10.4	<0.001
5. Cardiac Index L/min/m2	2.61±0.75	2.75±0.65	<0.05

Table-2

Immediate Hemodynamic results of BMV with single balloon technique (n = 23) and Double balloon technique (n = 9)

Parameter	Balloon	Pre dilatation	Post dilatation
Mitral valve area: (cm2)	Single Balloon	0.69±0.18	1.67±0.63
	Double Balloon	0.86±0.31	2.13±0.52
Mean Diastolic Gradient mm Hg	Single Balloon	16.2±5.8	4.7±3.3
	Double Balloon	13.6±4.9	3.0±1.5

Note: There was no significant difference in outcome whether single balloon or double balloon technique was used.

Table-3

Follow up Hemodynamic results after BMV in 24 patients

	Pre BMV:	Immediate Post BMV	Follow up
1. Mitral Valve area (cm2)	0.75±0.25	1.78±0.58	1.53±0.44
	P<0.001	P=NS	
2. Mean Diastolic gradient (mm Hg)	14.40±6.2	3.80±2.5	7.46±3.60
	P<0.001	P<0.001	
3. Pulmonary artery mean pressure (mm Hg)	36.00±13.4	28.00±11.3	21.50±5.20
	P<0.01	P<0.05	
4. Pulmonary artery systolic pressure (mm Hg)	54.00±19.3	43.80±16.6	35.00±9.10
	P<0.05	P<0.05.	
5. Cardiac Index ($L/min/m^2$)	2.53±0.59	2.71±0.67	3.08±0.71
	P<0.05	P<.05.	

Table - 4

Restenosis after BMV

Investigators	Patient No.	Mean age	Months follow up	Restenosis rate	mode of study
Palacious et al[10] (Echo Score<8)	27	49	13.0	4.0%	Catheterisation
Block et al[12]	41	52	24.0	51.0%	"
	41	52	24.0	16.0%	Echo
Abascal et al[13]	20	52	7.5	20.0%	Echo.
Vahanian et al[6]	91	43	9.0	4.0%	Echo.
Al Zaibag[14]	41	26	12.0	0.0%	Echo.
Desideri[11]	57	52	19.0	21.0%	Echo.
Present study	24	26	11.0	8.3%	Catheterisation.

Figure -1:

Functional class before and after BMV in 24 patients:

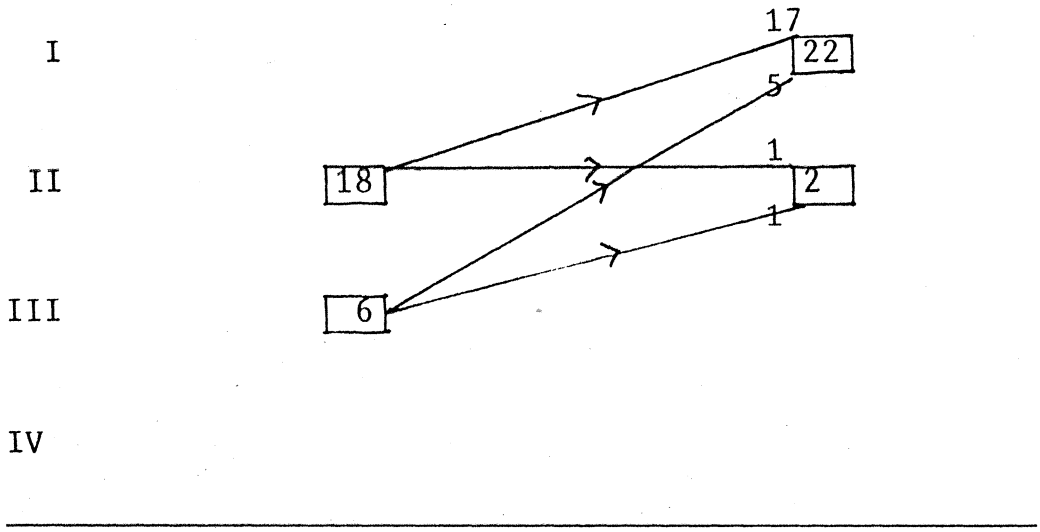
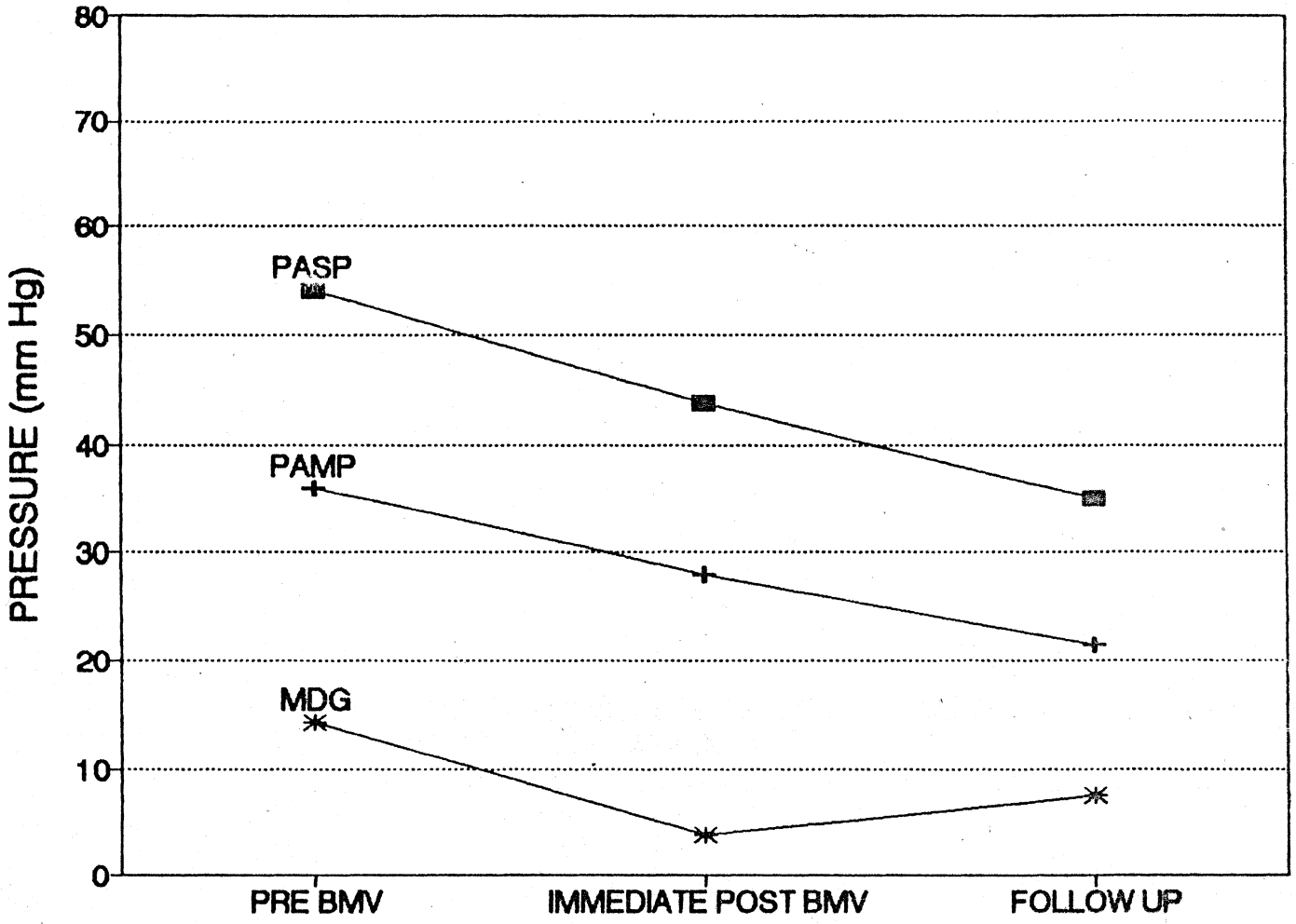


FIGURE 2: IMMEDIATE AND FOLLOW UP
RESULTS OF BMV IN 24 PATIENTS



PASP - Pulmonary artery systolic pressure
PAMP - Pulmonary artery mean pressure
MDG - Mean Diastolic Gradient

FIGURE 3: MITRAL VALVE AREA: IMMMEDIATE & FOLLOW UP VALUES IN 24 PATIENTS