CERTIFICATE

I, Dr.....G.JUSTIN PAUL.....hereby declare that I have actually carried out the two projects under report.

Signature.....

Place : Trivandrum

Date : 26.10.98

Name in capital letters

G.JUSTIN PAUL

Forwarded. He has carried out the two projects under report

Signature
Head of the Department

SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY TRIVANDRUM 695 011	Name	
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PROJECT WORKS DONE

1. **ACUTE PROCEDURAL RESULTS AND INTERMEDIATE TERM FOLLOWUP OF INTRA CORONARY STENTING (SCTIMST EXPERIENCE)**

2. **FIXED SUBAORTIC STENOSIS: A LONG TERM FOLLOWUP STUDY**

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SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES AND TECHNOLOGY TRIVANDRUM 695 011	Name	
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PROJECT REPORT

(PROJECT NO.1)

TITLE

ACUTE PROCEDURAL RESULTS AND INTERMEDIATE TERM FOLLOWUP OF INTRA CORONARY STENTING (SCTIMST EXPERIENCE)

NAME : **DR. G.JUSTIN PAUL**

PROGRAMME : **D M CARDIOLOGY**

MONTH & YEAR OF SUBMISSION : **NOVEMBER 1998**

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CONTENTS

ACUTE PROCEDURAL OUTCOME AND INTERMEDIATE TERM FOLLOW UP OF INTRA CORONARY STENTING (SCTIMST EXPERIENCE)

INTRODUCTION

Conventional balloon angioplasty introduced by Gruentzig et al in 1976 remains hampered by two vexing problems namely abrupt vessel closure during intervention needing emergency coronary artery bypass graft surgery and restenosis during follow up. Despite improvement in the operator skills, angioplasty materials, and a better understanding of the underlying pathologic process, the incidence of these complications have not decreased. Pharmacological therapy with direct acting thrombin inhibitors and glycoprotein IIb/IIIa inhibitors may prevent occurrence of these adverse outcomes in certain subgroup of these patients [1,2,3]. However bleeding problems are more frequent with some of these newer drugs.

The advent of intracoronary stenting was looked upon as an answer to most of the problems associated with conventional angioplasty.

Historical Overview

In 1968 Charles developed the first stents and used them in animals. In 1986 the first human intracoronary stents were implanted by Jacques Puell in Toulose, France and almost simultaneously by Ulrich Sigwart in Loussanne and Patrick Serruys in Rotterdam. In 1987 Gary Roubin and Douglas performed the first FDA approved intracoronary stenting in USA with GR Flex stents[4]. In 1988 the first Palmaz-Schatz stent was developed by Julio Palmaz and implanted by Richard Schatz [5]. In 1993 FDA approved the GR Flex stent for coronary use in acute and threatened closure.

Since then a multitude of stents of varying designs and properties have been developed and today's interventional cardiologist is at the luxury of picking and choosing the specific type of stent he prefers for a given type of lesion.

Evolution of the Technique

Goldberg [6] and Colombo [7] by means of intracoronary ultrasound demonstrated that stenting by conventional techniques was suboptimal in majority of the patients. In >80% of patients the stent expansion was unsatisfactory with inadequate apposition of stent struts to the vessel wall. They suggested additional high pressure dilatation using a noncompliant balloon under IVUS guidance for full expansion and complete apposition of stent struts to vessel wall. They achieved nil residual or even negative residual lesion by QCA using this technique.

The incidence of stent thrombosis [both acute and subacute] was very high in the initial periods. Hence the initial anticoagulant and antithrombotic protocol were vigorous leading to significantly higher bleeding complications associated with stents compared with conventional angioplasty. Subsequently Russo et al[8,9] performed optimal high pressure stenting under IVUS guidance and progressively diminished and finally stopped their post stenting anticoagulant regimen and observed very low stent thrombosis rates and bleeding complication rates with aspirin and ticlopidine alone. Soon efficacy of aspirin and ticlopidine in reducing stent thrombosis and bleeding complications were adequately demonstrated in many randomised controlled trials[10]. In many of these trials the use of intravascular ultrasound was not mandatory. Soon it was realised that with routine high pressure stenting, aspirin and ticlopidine,and with out intravascular ultrasound guidance similar good results could be achieved. The procedural success was very high and incidence of complications including stent thrombosis was very low[~1%].

So now in many centers intracoronary stenting is done with blind high pressure dilatation without intra vascular ultrasound and with aspirin & ticlopidine only with good results. This and the less steep learning curve of intracoronary stenting compared with other newer devices for percutaneous coronary interventions such as Rotoblator, Directional atherectomy etc., explains the current success of stents.

Landmark Trials

The use of intracoronary stents received a big boost with the publication of results of the BENESTENT-1 and STRESS-1 trials.[11 & 12]. These trials randomised patients with chronic stable angina with predominantly single vessel disease with mostly Type A AHA type lesions to conventional angioplasty and intracoronary stenting and observed that stenting significantly increased the procedural success and reduced the restenosis rate in angiographic follow up at an average of six months. The angiographic restenosis rates were 43% for ptca and 33% for stents in STRESS-1 and 32% for ptca and 22% for stents in BENESTENT-1 trials.

The BENESTENT-2 pilot study was a four phased study using heparin coated Palmaz –Schatz stents with progressive tapering of post interventional anticoagulation in each of the phases with finally stopping in phase IV. The overall restenosis rate was only 13%. The restenosis rate for phase IV where only aspirin and ticlopidine were used was as low as 6%[13].

Current Status of Stents

From the initial stage of stenting in single vessel disease in patients with chronic stable angina and non total occlusions, the technique has evolved now to

be available for almost all type of lesions and clinical situations. During the early periods it was felt that stenting in the thrombotic milieu of acute myocardial infarction will lead to very high incidence of thrombotic complications.However the recently published trials STENIM-1[14], PAMI-STENT[15], GRAMI[16], and FRESCO[17] have shown that stenting is not only safe but also more beneficial compared to conventional ptca in the setting of primary percutaneous intervention in acute myocardial infarction.

The SICCO trial and the SPACTO trial have shown the superiority of stents over ptca in the chronic total occlusions in improving acute procedural success and reducing restenosis during follow up. Similarly many trials have shown the benefit of using a coronary stent in various types of lesions [long lesions, short lesions, restenotic lesions[18], vein graft lesions, lesions with major side branches, bifurcation lesions, lesions in small vessels etc] and in variety of clinical situations[stable angina, unstable angina, Q and nonQ myocardial infarctions].

AIMS OF THE STUDY

1. To analyse the acute procedural results of the intracoronary stents done in the first two years in this institute eversince stenting was started.

2. To assess the intermediate term outcome of these patients in terms of restenosis.

3. To look for any patient related, lesion related or procedure related variable associated with restenosis.

MATERIALS AND METHODS

Study population

Intracoronary stenting was started in our institute in 2nd February 1996. All patients who underwent intracoronary stenting in our institute in the first two years from February1996 to January 1998 constituted the study population.

Clinical evaluation

Detailed coronary history was obtained from all patients including history of angina, unstable angina, and myocardial infarction {q and nonq}.Angina was classified according to the NYHA classification into class I to IV. Patients in whom stenting was done within one month of unstable angina were grouped as those with recent unstable angina and remote if they had unstable angina one month prior to stenting. Myocardial infarction was classified according to the territory involved in infarction {anterior, inferior, lateral, right ventricular etc.}.

Presence of coronary risk factors such as hypertension, diabetes, hyperlipidemia, smoking, family history of premature coronary artery disease, obesity etc were looked for. Noncoronary medical and surgical problems that are likely to influence the procedure/patient management like renal dysfunction, bronchial asthma, bleeding piles, acid peptic disease etc were also carefully looked for. Detailed clinical examination was made in all the patients. Chest x-ray, ECG, and echocardiogram were done in all the patients.

Indications for stent deployment

All patients had evidence of ischemia in the form of angina or inducible ischemia on treadmill stress test. Stents were deployed for the following reasons in our patients.

1. Elective : Primary aim was to reduce restenosis during follow up

2. Suboptimal ptca result : If there was a significant residual stenosis after ptca.

3. Bail out : When ptca resulted in acute or impending closure with dissections that could not be tacked up.

Procedure

Patients were taken to the cathlab. Arterial access was obtained usually with a single puncture technique from the right femoral artery using a 7 or 8 Fr sheath. A venous access was also obtained from the femoral vein. Most of our patients had a coronary angiogram done a few weeks prior to the procedure, though adhoc coronary stenting during diagnostic angiogram was done in a few patients. Diagnostic coronary angiograms were done in two orthogonal views to bring out the lesion to it's full extent with out fore shortening.

The length of the lesion, width of the narrowest point, width of the proximal and distal reference segments, % narrowing of the lesion etc were calculated by quantitative coronary angiography. The lesions were classified into type A, B1, B2, and C according to the AHA classification modified by Stephen Ellis et al.[19]. Presence of distal flow was classified into 0,1,2,and 3 according to the TIMI grading system. Presence of calcification if any was assessed during flouroscopy.

The lesion was usually crossed with a floppy guide wire. Intermediate and standard wires used in specific situations. Angioplasty was done with conventional rapid exchange balloon system,though over the wire balloons were used if needed. If significant calcium was seen in flouroscopy rotablation was done. After predilatation stents were deployed so as to cover the entire extent of the lesion. Both factory mounted stents and bare stents hand mounted in the cath lab were

used. Post deployment high pressure dilatation was done in most of the patients with a noncompliant balloon.

Anticoagulant/antithrombotic protocol

In the earlier part of our study we used aggressive anticoagulant protocol with 10,000 units of heparin bolus after arterial puncture with intermittent heparin to maintain ACT above 300 seconds. Heparin was continued at 1000 units per hour till the effect of oral anticoagulatns appeared. Oral anticoagulants were started on the evening prior to intervention and continued for three months. Sheaths were removed the next day.

Soon after the first few cases, we resorted to aspirin ticlopidine regime with ticlopidine being started two days prior to intervention and continued for six weeks after. Sheaths were removed in the same day six hours hours after the procedure unless otherwise indicated. Heparin was continued for 24 to 36 hours after the procedure. Patients were discharged usually on the third day of the procedure.

Follow up

All patients were advised to get regular blood counts done to watch out for neutropenia. They were called for a clinical evaluation and treadmill exercise testing at six weeks{when ticlopidine was stopped}and six months following the procedure. All patients were advised to be under the follow up of their local physician/ cardiologist also in the mean while.

Clinical restenosis was defined as recurrence of angina or evidence of inducible ischemia by treadmill exercise testing. Patients with clinical restenosis were advised to undergo coronary angiography. Angiographic restenosis was defined

as recurrence of narrowing at the stented site more than 50%. Overall restenosis was calculated by combining clinical and angiographic restenosis. While calculating overall restenosis rate, patients who have inducible ischemia by treadmill testing but no angiographic restenosis were excluded. The group with overall restenosis were compared with the group with out to look for any patient related, lesion related or procedure related variable that was associated with restenosis.

RESULTS

Baseline features

A total of 54 stents were deployed in 51 patients during the study period. 47 [92%] of them were male. The mean age was 52 + 8.26 years. Hypertension, diabetes mellitus and dyslipidemia were present in 17[33.3%], 16[31.3%], and 36[70.5%] patients respectively. 20[39.2%] patients were smokers and 15[29.4%] patients had family history of premature coronary artery disease.7 [13.6%] patients were obese.[Table-1]

37[72.5%] patients had exertional angina 33[64.7%] patients had NYHA class II angina and 4[7.8%] had class III angina.12[23.5%] patients had recent unstable angina and 8[15.7%] patients had remote unstable angina. 27 [52.9%] of our patients had atleast one prior myocardial infarction. 15[29.4%] patients had anterior wall myocardial infarction in the past and 9[17.6%] patients had inferior myocardial infarction. 11 [21.6%] patients had prior coronary revascularisation procedures done on them: 6 patients had a prior coronary angioplasty [4] of them were restenotic lesions] and 1 patient had a coronary artery bypass surgery done.[Table- 2].

None of our patients had significant left ventricular dysfunction.The mean ejection fraction was 68.6+8.6%.

Angiographic features

The distribution of AHA angiographic lesion subtypes A,B1,B2 and C in our patients is as follows: 13[24%], 17[31.5%], 14[25.9%] and 10[18.5%] lesions respectively. 27[50%] lesions were eccentric and 4[7.4%] lesions were restenotic lesions. 7[12.9%] lesions had sharp bends > 40 degree. 3[5.5%] lesions were

total coronary occlusions. 10[18.5%] lesions had atleast one major side branch from the lesion and 23[42.6%] had minor side branches from the lesion.[Table-3].

Overall 26[48%] lesions were in the proximal segments, 23[42.6%] lesions were in the mid segments and the rest in the distal segments of the coronary arteries. 36[66.6%] lesions were located in the LAD, 13[24%] in the RCA and 5[9.3%] in the LCX. [Table-4]

Indication for stenting

In 26[48.4%] lesions stenting was done on an elective basis with the primary intention of restenosis reduction. In 22[40.7%]lesions the indication was suboptimal result or poor luminal outcome with PTCA alone. Only 6[11.1%] lesions were stented for bailing out from acute and threatened closures. [Table-5]

Types of stents used

Palmaz Schatz [Johnson & Johnson] stent was the most commonly used stent,in 15[27.8%] lesions, followed by BE [Medtronic] stent in 10[18.5%] lesions. Wiktor stent in 7 [12.9%]lesions, Wiktor I and NIR stents were used in 6[11.1%] lesions each. GR II [Cook] stent was used in 5[9.3%] lesions. Crown [Johnson & Johnson] stent was used in 3[5.5%] lesions. Multilink[ACS] stent and Bard XT[Bard] stents were used in one lesion each.[Table-6].

Stent & lesion relation

The average lesion severity was 85.7 + 6.6%. The mean length of a lesion was 12.08+4.75 mm and the mean length of stent per lesion was 17.34+5.18 mm. The mean reference vessel dimension was 2.96+0.21 mm and the mean size of the stent used was 3.02+0.23 mm. The average stent/artery ratio was 1.02. 18 lesions

were located in vessels less than 3 mm reference diameter. 4 lesions were stented with 2.5 mm stents.[Table-7a &7b].

Procedure result

Procedural success as defined by the ability to deploy the stent in the desired position with less than 30% residual lesion was achieved in 49 patients [96.1%].. In two patients the stent could not be deployed in the desired location. In one patient the stent was retrieved back as it could not cross the proximal end of the lesion and in another patient the stent was deployed proximal to the desired site as the stent could not cross the distal end of the lesion. [Table-7a & 7b]

Most of our patients received post deployment high pressure dilatation. The mean inflation pressure was 12.2+1.98 atmospheres. The preprocedure lesion severity was 85.7+6.55% which came down to 4.5+7.2% post procedure. 7[13.2%] patients had undilated lesions of more than 70% severity. One patient had rotablation done with a 1.5mm burr. 15[28.3%] patients had undilated lesions of borderline significance.[Table-7a & 7b].

Complications

3 patients [5.8%] had a major side branch compromised and 6 patients [11.7%]had a minor side branch compromised. 6[11.7%] patients had a major bleed [defined as requiring atleast one unit of blood transfusion] and 7[13.2%] patients had a significant hematoma. 3[6%] patients had hemetemesis. 6[11.8%] patients had transient ST depression and 13 [25.5%] patients had transient T inversions. One patient each had transient atrial fibrillation, transient left bundle branch block, pulmonary edema requiring ventilation and nonoliguric renal failure. 9[17.6%] patients had non Q MI as defined by enzyme elevation with ECG changes. No patient had Q wave MI or needed emergency CABG.[Tables 7a,7b &8]

Follow up

All except 5 patients reported for the follow up clinical evaluation and treadmill exercise stress testing. One patient died 3 weeks after stenting due to subacute stent thrombosis. Excluding these 6 patients, all the other 45 patients came for the follow up treadmill testing after a period of 9.6 +5.12 months. The mean duration of follow up of our patients was 12.6+6.7 months.Clinical restenosis rate in our study was 37.8%[17 patients]. All these 17 patients had a treadmill test showing evidence of inducible ischemia. Two of them also had angina on exertion [Tables 9&10].

8 of the 45 patients[15.7%] underwent follow up coronary angiography of whom only two patient had angiographic restenosis[25%]. One patient subsequently underwent a coronary artery bypass surgery and the other patient is asymptomatic and is on conservative line of management. One patient had unstable angina 18 months after the procedure. Repeat coronary angiogram showed no restenosis of the target lesion. However an initial mild circumflex lesion had progressed.

Three patients who underwent coronary angiography for clinical restenosis did not have angiographic restenosis. Excluding these three patients the overall restenosis rate was 31.1%[14 patients]. The distribution of the various patient related, lesion related and procedure related variables between the two groups are shown in the tabular columns 11,12 and 13. We found that overall restenosis had exclusively occurred in patients with LAD lesions. In other words none of our patients with stenting done to RCA and LCX lesions had overall restenosis. In calculating restenosis rates for subgroups we found that diabetics and patients with proximal LAD stenting had very high incidence of restenosis{53.8% and 50% respectively [Tables 9 to 14]. The distribution of various types of stents implanted and their restenosis rates are given in Table 15.

DISCUSSION

The age and sex distribution in our patients is like the usual distribution of age and sex in coronary population. The youngest patient in our study was 37years old and the oldest patient was 72 years old. The prevalence of coronary risk factors in our study population was similar to that in other studies except that dyslipidemia was more prevalent in our patients[72%].

Though the study included the patients who were stented in the first two years since it was started in our Institution, most of our patients had complex lesion morphology. 58.4% of our patients had type B lesions and 19% had Type C lesions. Only 22.6% had simple Type A lesions. This is in contrast to BENESTENT and STRESS trials which contained predominantly Type A lesions. The mean lesion length in BENESTENT trial was 7 mm, while it was about 12 mm in our study[11,12].

Despite having more complex lesions, our 96% procedural success was quiet impressive and is comparable to any other trial elsewhere. Elective stenting and stenting for suboptimal ptca outcome were the commonest indications for stenting in our study [47% and 41.5% respectively]. Only 11.3% were stented for bail out indications.

Slotted tube stents were the commonest stents used in our patients[66%]. All the rest received coil stents except one who received a hybrid type stent. The slotted tube stents have a greater radial strength, but their trackability is not very good. The coil stents have a better trackability at the cost of their radial strength. The hybrid design stents were designed to have good radial strength as well as good trackability. The commonest stent used in our study was Palmaz Schatz stent from Johnson & Johnson., the stent that has been extensively studied in many

randomised controlled trials like BENESTENT, STRESS etc. Radio-opacity is a feature of stents that help the operator in precise placement of the stents. Of the stents we had used radio-opacity of Wiktor and Wiktor I were good, and that of GRII and Bard XT were fair enough. All other stents we used[Palmaz Schatz, Medtronic Be, NIR, Crown, ACS Multilink] had poor radio-opacity.

Subacute stent thrombosis

IVUS performed immediately after implantation of stent demonstrated that despite angiographic appearance of complete stent expansion, most stents are in fact inadequately deployed by traditional balloon inflation pressures[6 to 8 atmospheres] with poor apposition of stent struts to the vessel wall[20] . It was postulated that stent thrombosis primarily arose at the site of poorly supported arterial plaques or where the stent struts protrude into the arterial lumen. Subsequently it was shown that high pressure dilatation under intravascular ultrasound guidance ensures adequate stent strut apposition[7,8]. Almost all our patients received high pressure dilatation. However we did not use intravascular ultrasound in any of our patients. Recently studies have shown that routine use of intravascular ultrasound may not be necessary and blind high pressure is good enough to ensure adequate strut apposition [21,22].

Although subacute stent thrombosis was substantially higher in the early experience, the employment of routine high pressure dilatation in the recent studies have reduced this complication to about 1% despite reduced anticoagulation.[23]. The incidence of stent thrombosis was 3.4% and 3.5% in BENESTENT-I and STRESS-I respectively[11.12]. The incidence of stent thrombus in our study was 1.9%[one patient]. This patient a 72 year old lady who was a poor risk candidate for coronary artery bypass graft surgery in view of diffusely diseased coronaries

and coexisting general medical problems, had a long stent implanted covering proximal and mid left anterior descending artery. She had a good result procedure in terms of < 10% residual narrowing and no major procedural complications. However the patient died suddenly after three weeks, the cause of which we presume to be subacute stent thrombosis. Long stents have been shown to be associated with higher risk of stent thrombosis by Schatz et al.[24].

Antithrombotic Protocol

During the initial phase of our study we followed intensive anticoagulant and antithrombotic protocol with aspirin, ticlopidine, heparin and oral anticoagulation. The incidence of major bleed was higher in our patients[11.7%]. However most of the bleeding complications occurred during our early intensive anticoagulation regimen. The incidence of major bleed in BENESTENT and STRESS trials were 13% and 7.3% respectively [11,12]. These trials had also used intensive anticoagulation regimen with Heparin, warfarin, aspirin and ticlopidine. BENESTENT continued warfarin for 3 months and STRESS for one month only. We continued oral anticoagulation for 3 months.

Subsequently when we switched over to aspirin /ticlopidine regimen with heparin only during the first 24 to 36 hours, major bleed incidence came down to 4.4%.Kasrati et al in the ISAR trial showed that aspirin and ticlopidine given for a period of four weeks after the procedure was associated with significantly reduced acute ischemic complications [7]. Newmann et al showed that aspirin alone was inferior to combined aspirin and ticlopidine in suppressing platelet activation after intra coronary stenting [25].

The incidence of ticlopidine induced neutropenia has been reported to be about 1 ~ 2% [26] . However we did not have any patient with significant neutropenia neccessitating stopping of ticlopidine.

Restenosis

Restenosis is the Achilles heel of ptca. The mechanisms of ptca restenosis include recoil, thrombus formation and organisation, vessel wall remodeling and neointimal proliferation. Intra coronary stenting takes care of recoil and vessel wall remodeling. Neointimal proliferation remains the primary mechanism of instent restenosis. The rate of instent restenosis has been reported to be as low as 6% in phase IV of BENESTENT –I trial and 7% in Multicenter Stenting [MUST] [27] study to 33% in STRESS-I trial and 37% in TASTE [Ticlopidine Aspirin STent Evaluation] [28]study. Studies done in patients with high risk for restenosis like chronic total occlusion etc had still higher restenosis rates. Our angiographic restenosis rate was 25%, which is similar to what is reported elsewhere in the literature. However only15.7% of our follow up patients underwent follow up angiography. Hence this will not reflect the true angiographic restenosis of our study. Our overall restenosis rate combining clinical evaluation, treadmill evaluation and angiography was 31.1% which again is comparable to what is reported elsewhere in the literature.

Satler et al and others found that diabetes, unstable angina, restenotic lesions, smaller reference vessel diameter, smaller final lumen diameter, chronic total occlusions etc were predictive of restenosis [29]. We compared the group with over all restenosis with the group without restenosis and found that of the known coronary risk factors hypertension, dyslipidemia, smoking and family history of premature coronary artery disease were almost equally distributed. However diabetes was more prevalent in the restenosis group {50% Vs 19%}. LAD lesions were more common among the restenosis group {100% Vs 58%}. The distribution of other lesion specific and procedure related variables were almost the same in both

the groups. The overall restenosis calculated for the individual subgroups was highest for the diabetic group {53.8%} followed by LAD subgroup {50%}. LAD lesions have been reported to be associated with a higher incidence of restenosis.

Although there was an initial euphoria that stenting will take care of the problem of restenosis, it was soon realised that it could only tackle a smaller part of the problem of restenosis. However with the advent of newer innovations like stents coated with genetically engineered endothelial cells, endovascular brachy therapy etc it is likely that in the days to come the problem of restenosis can be completely taken care of.

CONCLUSIONS

1. Intra coronary stenting is a viable option for management of patients with ischemic heart disease
2. Intracoronary stenting can be done with a very low incidence of major complications.
3. Acute procedural results are good with less than 5% residual lesion in most of the cases.
4. Instent restenosis is still a potential problem with intra coronary stenting.
5. Diabetes and proximal LAD lesions are associated with a higher incidence of restenosis.

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Table 1
BASELINE DATA

	No.	%
No. of Patients	51	100
Male	47	92
Female	4	7.8
Hypertension	17	33.3
Diabetes	16	31.3
Dyslipidemia	36	70.5
Smoking	20	39.2
Fam H/o CAD	15	29.4
Obesity	7	13.7
Mean EF in %	65.56	± 8.63
Mean age in years	52	± 8.26

Table 2
CORONARY HISTORY

	No	%
CSA Class 2	33	64.7
CSA Class 3	4	7.8
Past unstable angina	8	15.7
Recent unstable angina	12	23.5
MI	27	52.9
Non Q MI	6	11.7
AWMI	15	29.4
IWMI	9	17.6
Post PTCA	6	11.8
Post PTCA [Restenosis]	4	7.8
Post CABG	1	2

Table 3
LESION CHARECTERISTICS

	No	%
Type A	13	24
Type B1	17	31.5
Type B2	14	25.9
Type C	10	18.5
Eccentric	27	50m
Concentric	27	50
Restenotic	4	7.4
Bend > 40°	7	12.9
Bend ~ 20-40°	5	9.3
Total Occlusion	3	5.5
Major Side Branch	10	18.5
Minor Side Branch	23	42.6

Table 4
LESION LOCATION

	No	%
Total No. of Lesions	54	100
LAD	36	66.6
LCX	5	9.3
RCA	13	24
Proximal Lesions	26	48
MID Lesions	23	42.6
Distal Lesions	4	7.5
Proximal Lad	22	40.7
Mid Lad	14	25.9
Proximal LCX	3	5.6
Distal LCX	2	3.7
Proximal RCA	2	3.7
Mid RCA	9	16.6
Distal RCA	2	3.7

Table 5
INDICATIONS FOR STENTING

	No	%
Elective	26	48.4
Poor Luminal Outcome	22	40.7
Bail out	6	11.1

Table 6
TYPE OF STENTS USED

	No	%
Total No. of Stents	54	100
PSS	15	27.8
Med. Be	10	18.5
Wiktor	7	12.9
Wiktor I	6	11.1
NIR	6	11.1
GR II	5	9.4
Crown	3	5.5
ACS Multilink	1	1.8
Bard XT	1	1.8

Table 7a
PROCEDURE DETAILS

	Average	Std. Devi.
Lesion Length	12.08	4.75
Stent Length	17.34	5.18
Vessel size	2.96	0.21
Stent size	3.02	0.23
Stent / Artery Ratio	1.02	0.02
Max.Deployment Pr. (in atm. pr.)	12.2	1.98
Lesion Severity in %	85.7	6.55
Final Residua in %	4.5	7.2

Table 7b
PROCEDURE DETAILS

	No	%
Procedures success	52	96.3
Vessel Size < 3 mm	18	33.30%
Stent Size ~ 2.5 mm	4	7.40%
Side br. Compromise [Major]	3	5.8
Side br. Compromise (Minor)	6	11.7
Undilated lesions >70%	7	13.2
Undilated lesions 50-70%	15	28.3

Table 8
COMPLICATIONS

	No.	%
Major Bleed	6	11.7
Hematoma (significant)	7	13.7
Transient ST Depression	6	11.8
Transient T wave changes	13	25.5
Transient AF	1	1.9
Transient LBBB	1	1.9
Non Q MI	9	17.6
Hemetemesis	3	5.8
Angioedema/Contrast Reaction	2	3.9
Pulmonary Edema	1	1.9
Non oliguric renal failure	1	1.9
Death in Hospital	0	0

Table 9
FOLLOW UP DATA

	No	%
Treadmill Followup	45	88.3
ReCAG. Done	8	15.7
Stent Thrombosis	1	1.9
Death in Followup	1	1.9
Emergency CABG	0	0
Elective CABG	1	1.9
Unstable Angina	2	3.9
Exertional Angina	1	1.9
Repeat PTCA	0	0
T.L.R	1	1.9
Lost in Followup	5	9.8

Table 10
FOLLOWUP DATA [45 PATIENTS ONLY]

	No	%
Clinical restenosis	17	37.8
CAG restenosis	2	25
Overall restenosis	14	31.1
Time in Months	**Average**	**Std.Devi.**
Net F.up Duration (Months)	13.4	6.7
TMT F.up Duration (Months)	10.2	5.1

Table 11
OVER ALL RESTENOSIS ANALYSIS

Variable	Restenosis group (n=14)	No Restenosis group (n=31)
Clinical History	Number (%)	Number (%)
Hypertension	4 (28.6)	10 (32.2
Diabetes	7 (50)	6 (19.4)
Dyslipidemia	9 (64.3)	24 (77.4)
Smoking	6 (42.9)	15 (48.4)
Family History	5 (35.7)	7 (22.6)
H/O MI	9 (64.3)	14 (45.2)
Recent UA	4 (28.6)	7 (22.6)

Table 12
OVER ALL RESTENOSIS ANALYSIS

Variable	Restenosis group (n=14)	No Restenosis group (n= 31)
Lesion site / type	Number (%)	Number %
LAD	14 (100)	18 (58)
Prox LAD	10 (71.4)	10 (32.)2
Mid LAD	4 (28.6)	8 (25.8)
RCA	0 (0)	10 (32.2)
LCX	0 (0)	3 (9.6)
Bend > 40°	2 (14).3	3 (9.6)
Eccentric	6 (42.)9	13 (41.9)
Type A	2 (14.3)	10 (32.2)
Type B1	6 (42.9)	9 (29)
Type B2	4 (28.6)	6 (19.4)
Type C	2 (14.3)	6 (19.4)
Total occlusion	1 (7.1)	2 (6.4)
Restenotic Lesion	1 (7.1)	1 (3.2)

Table 13
OVERALL RESTENOSIS ANALYSIS

Variable	Restenosis group (n=14)		No.Restenosis group(n=31)	
	Mean	Std.Devi.	Mean	Std.Dev.
Ejection Fraction (%)	65	7.3	66.4	7.1
Lesion Length (mm)	12.1	3.5	11.4	4.1
Stent Length (mm)	16.4	3.1	17.2	4.6
Vessel Size (mm)	2.97	0.25	2.96	0.2
Stent Size (mm)	3	0.28	3	0.2
Lesion Severity (%)	87	6.4	84.7	6.9
Max. Atm. Pr.used	12.9	2.2	12.2	1.8
Final Residua (%)	3.6	8.4	4.1	7.3

Table 14
OVERALL RESTENOSIS ANALYSIS

VARIABLE	Net	Restenosis	% Restenosis
No. of Patients	45	14	31.1
LAD	32	14	43.8
Proximal LAD	20	10	50
Mid LAD	12	4	33.4
Diabetes	13	7	53.8
F/H of CAD	12	5	1.7
Smoking	21	6	28.6
Dyslipidema	33	9	27.3
Hypertension	14	4	28.6
H/O recent UA	11	4	36.4
H/O MI	23	9	39.1
Type A	12	2	16.7
Type B	25	10	40
Type C	8	3	40

Table 15
OVER ALL RESTENOSIS ANALYSIS

Type of Stent	No.Implanted	Restenosis	% Restenosis
Palmaz Schatz	13	3	23
Wiktor	12	2	16.6
Medtronic Be	10	6	60
NIR	5	1	20
GR II	3	0	0
Crown	3	2	27
Multilink	1	0	0

PROJECT REPORT

(PROJECT NO.2)

TITLE

FIXED SUBAORTIC STENOSIS : A LONG TERM FOLLOWUP STUDY

NAME : **DR. G.JUSTIN PAUL**

PROGRAMME : **D M CARDIOLOGY**

MONTH & YEAR OF SUBMISSION : **NOVEMBER 1998**

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CONTENTS

FIXED SUBAORTIC STENOSIS :
A LONG TERM FOLLOW UP STUDY

Introduction

Left ventricular outflow obstruction can occur at three different levels: valvar, subvalvar and supravalvar. Subvalvar obstructions can be fixed or dynamic. Fixed subaortic stenosis [FSAS] is an uncommon anomaly in which left ventricular outflow tract [LVOT] is narrowed by various mechanisms alone or in combination [1]. The first discription of FSAS is attributed to Chevers in 1942[2]. Berth reported results of trans ventricular dilatation as treatment in 1956[3]. Tunnel form of FSAS was described by Spencer et al in 1960[4] and later reemphasised by Morrow et al [5]. Aortoventriculoplasty was introduced by Raston et al in 1975 and modified Konno operation was introduced in 1978, for surgical corrrection of FSAS.

Fixed subaortic stenosis is the second most common form of fixed LV outflow obstructions [6]. In about 20% of patients with fixed LVOT obstructions, the level of obstruction is subaortic[7]. It is two to three times more common in males. Unlike valvar aortic stenosis this condition is almost never seen in new borns and rarely seen in infants. While the substrate or matrix of these forms must clearly be present in the newborn, it is extremely unusual for it to be of any clinical relevance in the first few months of life [8]. It is thought that FSAS is an acquired lesion with an ill-defined underlying congenital pathology [6]. It usually occurs as a critical lesion after the age of 5 years or later, and most commonly after 10 years of age. Familial occurrence has been reported [6,10,11,12].

Morphology

FSAS can be caused by a thin discrete fibrous membrane [75 – 85%], a thick fibromuscular incomplete diaphragm [5 – 10 %], or a diffuse tunnel like narrowing of the LVOT [5-10 %][6]. The obstruction can be located anywhere from the nadir of the aortic cusps to the free edge of anterior mitral leaflet and anywhere along the aorto-mitral annulus. A high ridge may be attached to the aortic valve [right coronary cusp only, or all the three cusps] and a low ridge may be attached to the hinge line of anterior mitral leaflet. The commonest type of FSAS is a crescent or less commonly a complete ring of fibroelastic tissue, which lies about 5 to 10 mm below the aortic valve. In transposed great arteries FSAS lies below the pulmonary valve [7].

The tunnel type presents as an irregular area of fibrosis commencing close to the aortic annulus and extending downwards for about10 to 30 mm. This may have varying degree of severity of narrowing. In the most severe form the tunnel is long and aortic annulus is small sized, though the aortic valve leaflets as such are normally formed. In the milder form the tunnel may be short and annulus normal sized when the morphology tends to resemble the fibromuscular form. Transformation of discrete FSAS to tunnel type is rare in untreated patients, but has been reported after surgical resection [13].

The diameter of the aortic annulus is on an average smaller than normal[14]. Aortic valve is usually tricuspid. Trivial or mild aortic regurgitation is present in 66% of the patients at diagnosis. Aortic valve however can be bicuspid with varying degrees of commisural fusion producing associated valvar aortic stenosis. The base of the aortic valve cusp is thickened when a high lying ridge is continuous with it. Acquired jet lesions of the aortic valve leads to an abnormal aortic valve in almost every patient after the age of 5 years [7]. Aortic valve can be damaged by infective

endocarditis, a not uncommon complication of FSAS. Unlike valvar aortic stenosis, ascending aortic dilatation is uncommon though rarely it can occur [7]. Mitral valve abnormalities occur in about 10% of patients. Anterior mitral leaflet can get restricted in motion and thickened and can lead to mild degrees of mitral regurgitation. Mitral regurgitation can also occur due to severe LV hypertension and papillary muscle dysfunction, mostly due to combination of these factors [9].

The left ventricle is usually concentrically hypertrophied. Subendocardial ischemia and fibrosis can occur. Rarely there is excessive hypertrophy of the inter ventricular septum more than the posterior wall, when differentiating from hypertrophic cardiomyopathy may be difficult. Roberts et al [15] have noted the presence of coronary luminal narrowing due to structural wall changes in the intramural coronary arteries. Coronaries may appear dilated in some patients[9].

Associated Lesions

Associated lesions are more common than valvar aortic stenosis and they occur in about 50 to 65% of patients [6]. Aortic valve can be congenitally abnormal in 20-25% of patients with FSAS, however in majority of the patients the abnormality in the aortic valve is secondary to jet effect as we had seen earlier [6]. Secondary damage to the aortic valve may emerge as the predominant lesion in untreated patients. Other commonly associated anomalies include ventricular septal defect [VSD], patent ductus arteriosus, coarctation of aorta and atrioventricular septal defects. Valvar and infundibular pulmonary stenosis[16], double chambered right ventricle[17], aorto-pulmonary window, aneurysm of sinus of valsalva[18], aneurysm of membranous interventricular septum, tetrology of Fallot etc have been reported to coexist.

Parachute mitral valve, supramitral ring and coarctation of aorta can occur as a part of Shone's complex [19]. Diffuse subaortic stenosis occurs in patients with interrupted aortic arch and in severe coarctation of aorta as a part of 'Becu Complex,' where abnormal muscle bundles are located in the LVOT. Complex type of FSAS occurs in patients with discordant atrio-ventricular and ventriculo-arterial connections. Patients with single ventricle, double outlet right ventricle[20], d-transposition of great arteries and anatomically corrected malposition can have associated subaortic narrowing. The other uncommonly associated anomalies include Ebsteins anomaly[21], hypertrophic cardiomyopathy[22], anomalous coronary origin from pulmonary artery[23], single coronary artery[24] etc.

Pathogenesis

Rosenquist noted greater aorto-mitral discontinuity in patients with FSAS compared with normal population due to a muscular wedge between aortic and mitral annuli [14]. Though the morphologic basis of this is uncertain it is possible that abnormal flow patterns associated with this abnormality could lead to abnormal proliferation of tissue[6] in this area obstructing the LVOT[9]. Van paraag et al in 1971 suggested that while anatomical potential for subaortic stenosis is present at birth, hemodynamically significant LVOT obstruction is usually acquired post natally[9]. The acquired nature of the lesion is suggested by recurrence of identical tissue after surgical removal[7]. The fact that FSAS has been found to develop after resection of coarctation of aorta, spontaneous[25] or surgical closure of ventricular septal defects, pulmonary artery banding [26], surgical correction of atrioventricular septal defects[27],surgical correction of tetralogy of Fallot[28] and replacement of left AV valve, goes to prove the acquired nature of the disease.

Persistent embryological endocardial cushion tissue that retains its proliferative capacity has been suspected to be responsible[6]. The view of Kieth et al that FSAS results from incomplete resolution /atrophy of bulbous cordis still remains speculative.[9]. Abnormally placed anterior mitral leaflet could also lead to FSAS.

When a VSD is associated with FSAS the pathogenesis is variable depending on whether the VSD is above or below the stenosis. When the infundibular septum goes for a posterior and caudal malalignment encroaching into the LVOT the morphological basis is for FSAS above the VSD. This is the most common type of FSAS found in children below one year of age. In certain congenital heart disease this malaligned infundibular septum may hypertrophy in response to a pulmonary artery banding leading to subaortic obstruction. So Van paraag suggests that such posterior malalignment when documented by angiography should contra indicate a pulmonary artery banding.

Most fibromuscular obstructions are located below the VSD. Here the subaortic obstruction may be due to accessory mitral/tricuspid tissue tags, anomalous muscle bundle in LV, malattached mitral valve etc. A restrictive bulboventricular foramen in single ventricle with transposed great arteries, a restrictive VSD in mitral atresia with normal aortic root, malattached superior bridging leaflet in AV canal defects, poorly expanded subaortic infundibulum in anatomically corrected malposition, anomalous LV muscle bundles in 'Becu complex'[7], herniation of tricuspid valve tissue thro a small VSD [7], etc contribute to potential subaortic obstructions.

Natural History

The natural history of FSAS is influenced by the high incidence of associated anomalies. Congestive cardiac failure and sudden cardiac death seen in some young patients are often due to associated anomalies. In majority of patients FSAS is progressive. About 75% of patients have an increase in gradients of >25% in 5 years period {more rapid than valvar AS}[6]. Rapid progression is more common in tunnel narrowing. Incidence and severity of AR increases with age. Incidence of bacterial endocarditis is reported to be as high as 13 to 25%[6]. Sudden cardiac death has been reported, but exact incidence is uncertain.

Clinical Features

In young patients, clinical features are frequently masked by those of the associated lesions. In grownups giddiness and effort syncope occur more frequently than valvar aortic stenosis. Dyspnea is a common complaint. Patients can present with typical effort angina. They may rarely present with infective endocarditis. A systolic murmur is heard but a click is rare(present in 5%) [6]. There is an audible early diastolic murmur in atleast more than two third of the patients. When the stenosis is severe, the pulse is slow rising, the second heat sound is single or paradoxically split and a fourth heart sound may be audible at the apex.

Heart size is usually normal. Ascending aorta is not usually dilated in isolated FSAS though it may be dilated in about 20% of the patients. Valvar calcification is absent. The ECG shows left ventricular hypertrophy [LVH]. Upto 65 to 85% have LVH and about 25% have LVH with strain pattern. Infants are more likely to show right ventricular hypertrophy.

M Mode examination shows the abnormal fluttering of aortic valve during systole and early systolic closure of the aortic valve. 2D echo is diagnostic and demonstrates the site, thickness and extent of the FSAS and shows the distance of the FSAS from the aortic cusps. It can demonstrate the presence or absence of aortomitral discontinuity. It will show the extent of involvement of the aortic and mitral valve tissues by the subaortic ridge/membrane. Continuous wave doppler can asses the LVOT gradient. Color doppler evaluation can be used to quantify the associated aortic regurgitation.

Cardiac Catheterisation and Angiography

On cardiac catheterisation there is a systolic pressure gradient below the aortic valve on careful withdrawl of a catheter with closely spaced sampling holes across the left ventricular outflow tract. Post ectopic response is normal unlike hypertrophic cardiomyopathy. LV entry is easier than in patients with valvar aortic stenosis. LVOT is best profiled in long axial projection and elongated RAO projection. In patients with aortic regurgitation the subaortic chamber is sometimes well visualised in aortic root angiography.

Treatment

Excision / enucleation of the subaortic ridge/membrane under vision, through carefully retracted aortic valve using cardiopulmonary bypass is the treatment of choice. It is important to remove as much as membrane as possible without damaging the mitral/aortic valves and without damaging the bundle of His. Operative mortality has been reported to be around 0 to 6%. Tunnel form has a higher operative mortality. Subaortic stenosis may recur and may need redo surgery. Lupinetti et al showed the benefits of doing a myomectomy along with membrane resection in terms of reduced need for redo surgery [29]. Similar findings were reported by Lavee et al [30].

Surgery may be needed for associated aortic regurgitation which may be aortic valve repair/replacement. Mitral valve repair/replacement may be needed for the associated mitral valve anomalies.

Many authors recommend early surgery as soon as the diagnosis is made, even in the absence of substantial subaortic pressure gradients, aortic regurgitation or left ventricular hypertrophy. However since postoperative follow up reports have only described patients who have been operated in the advanced stages of the disease, the benefits of early surgery has not yet been proved.[31-35]

AIMS

1 To analyse the long term follow up of operated and not operated patients with a diagnosis of Fixed SubAortic Stenosis [FSAS].

2 To look for the associated lesions of the fixed sub aortic stenosis.

3. To see if the distance of the FSAS from the aortic valve correlates with the progression of left ventricular outflow tract [LVOT] gradient.

MATERIALS AND METHODS

All patients who were diagnosed to have Fixed Subaortic Stenosis in our institute between 1980 and 1996 were included in this study. The case sheets of all the patients in the medical records department were reviewed. The clinical history, physical examination, ECG, chest x-ray, echocardiographic and cardiac catheterisation records were reviewed. All cine angiograms done were reviewed. Only patients who satisfied the following inclusion criteria were included.

1. The patients should have had atleast one detailed cardiac catheterisation study.
2. The patients should have had detailed follow up evaluation spanning over a period of atleast two years.
3. Patients should have concordant atrioventricular and ventriculoarterial connections.

The following patients were excluded from the study

1. Patients with obstruction of the LVOT caused by hypertrophic cardiomyopathy.
2. Patients with complex type of subaortic stenosis with major associated anomalies like univentricular heart, transposition of great arteries, double outlet right ventricle, tetrology of Fallot, interrupted aortic arch etc were excluded. [However patients with the usual associated lesions of FSAS like coarctation of aorta, ventricular septal defects, patent ductus arteriosus, right ventricular outflow tract obstructions etc were included.]

3 Patients with insufficient echocardiographic data and/or inadequate follow up data were excluded.

The criteria for diagnosis was the demonstration of a localised subvalvar diffuse ridge or long segment narrowing in the outflow tract. The diagnosis was established by echocardiography, cardiac catheterisation, angiography, and direct visualisation during surgery. The lesions were defined as discrete (short segment) or tunnel type (long segment). Patients in whom the length of the obstruction was more than one third of the aortic annulus were classified as tunnel type [36]. The distance of the subaortic ridge/membrane from the aortic valve cusps was available in all our patients.

The patients were divided into two groups depending on whether they were operated for FSAS or not : Group I – operated for FSAS, Group II- not operated for FSAS or operated for some associated lesions only. Group II patients were either awaiting surgery or they were not operated for, as the FSAS was felt less significant or for socioeconomic reasons. The patients were further subdivided depending on the distance of the subaortic membrane from the aortic valve cusps into two subgroups A: distance < 6mm, and B: distance ≥ 6mm.

RESULTS

38 patients who met the study criteria were included in the study protocol. Sixteen of them were females and twenty two were males { Table-1}. The mean age at registration was 12 ± 8 years{ range 4 to 34}.4 patients were asymptomatic at presentation. Dyspnea on exertion was the commonest complaint and was present in 32[84%] patients. 8 patients [21%] had angina and 2 patients [5.3%] presented with syncope. Two patients [5.3%] in our series had infective endocarditis some time in the course of the disease. Two patients presented with congestive heart failure and these patients had a large VSD or a large ductus shunting left to right. One patient had an attack of rheumatic fever after registration with us {Table-2}.

Clinical examination revealed ejection click in two [5.3%] and ascending aortic dilatation in three [26.3%] patients. Electrocardiography revealed LVH in 36 patients [94.7%] and strain pattern in 18 patients [47.4%]. Chest X-Ray revealed cardiomegaly in 8 patients [21%].Three patients [7.9%] had tunnel type of subaortic narrowing and the 35 remaining patients[97%] had discrete FSAS. The mean distance of the sub aortic membrane from the aortic valve was 6.8 ± 2.8 mm {Table-3}.

All patients underwent cardiac catheterisation at a mean age of 15.6 ± 8.5 years {range 7 to 36}. The LVOT gradient during catheterisation was 88.5 ± 52.9 mm of Hg. 26 patients had angiographically demonstrable aortic regurgitation [mild in 21 patients, moderate in 5 patients and severe in none]. Seven of them had undergone a repeat cardiac catheterisation later in the follow up.

Associated anomalies

26 patients [68.4%] had associated anomalies while 12 patients [31.6%] had isolated FSAS {Table-4}. Ventricular septal defect was the commonest anomaly and was found in 9 patients [23.7%] and all were of perimembranous variety. Patent ductus was the next common associated anomaly found in 8 patients [21.1%]. Mitral valve regurgitation was found in 5 patients [13.2%] and mitral stenosis was found in 3 patients [7.9%]. The cause of mitral stenosis was supramitral ring in two patients [5.3%] and single parachute papillary muscle in one patient. Of the 5 patients with mitral regurgitation one had a cleft anterior mitral leaflet and one had a cleft posterior mitral leaflet. Four patients [10.5%] had significant pulmonary hypertension. All these four patients had significant left to right shunt at either ventricular or great arterial level. Three patients [7.9%] each had bicuspid aortic valve and coarction of aorta. Two patients [5.3%] each had coarctation of aorta, infundibular pulmonary stenosis and double chambered right ventricle. One patient each had valvar pulmonary stenosis, single coronary artery, coronary artery aneurysm, aneurysm sinus of valsalva, aortopulmonary window, interrupted inferior venacava, atrial septal defect, membranous septal aneurysm with out ventricular septal defect, and left ventricular dysfunction. One patient had rubella syndrome and one patient had down's syndrome.

Balloon Dilatation

Three patients underwent balloon dilatation of subaortic membrane {Table-5}. Though the gradients came down in all the three patients, the residual gradients at the end of the procedure was > 40 mm of Hg in all of them. Two of them got operated for FSAS. The third patient was advised surgery however patient was not willing for surgery.

Surgical Procedures Done

Of the 38 patients 28 were operated {Table5,6}. The mean age at surgery was 18.3 ± 8.8 [range:9 to 39] years. Of this 28 patients, 22 patients were operated for FSAS, and the remaining patients were operated for associated lesions only. The 22 patients operated for FSAS formed Group I, and the remaining 16 patients not operated for FSAS formed Group II. Among the Group I patients in addition to removal of FSAS 3 patients had replacement of mitral valve and 2 patients had replacement of aortic valve. 5 patients had additional myomectomy/myotomy done.

The other procedures done in our patients include VSD closure in 6 patients, PDA ligation in 7 patients, coarctation repair in 3 patients, infundibular resection in 2 patients, and ASD closure, sinus of Valsalva repair, post coarctation surgery aortic false aneurysm repair, Kay's annuloplasty of mitral valve, mitral leaflet cleft repair, aortic valve commisurotomy in 1 patient each.

Mortality

Three patients died after surgery. One patient who underwent FSAS excision had a small aneurysm of noncoronary cusp of aortic valve, and this was left untouched during surgery. He presented 7 years later with a pulsating mass protruding through sternum. He was found to have a large friable aneurysm of noncoronary cusp of aortic valve during surgery, and he died of intractable bleed while trying to repair the aneurysm. The second patient died of sudden unexplained hypotension in intensive care unit 6 hours after an apparently successful surgery. The 3rd patient presented with infective endocarditis 2 months after surgery and she died of multiorgan system failure.

Aortic regurgitation

At the time of diagnosis 27 of the 38 patients[71%] had echocardiographically documented aortic regurgitation {Table-7}. 23 of them had mild regurgitation, 3 of them had moderate regurgitation and one had severe regurgitation. The findings in cardiac catheterisation were almost the same with angiographically demonstrable aortic regurgitation in 26 patients [mild in 21 and moderate in 5]. At the time of last follow up 31 of the 38 patients[81%] had aortic regurgitation, mild in 13 patients, moderate in 15 patients and severe in 3 patients. New regurgitation had developed in 4 patients in whom it was not present at time of diagnosis. It had worsened by one grade in 15 patients, two grades in 4 patients and had regressed by one grade in 3 patients.

Subgroup analysis: operated Vs non-operated

In our Group I patients, the LVOT gradient at the time of diagnosis was 81.4 ± 37.8mm of Hg {Table-8}. The gradient obtained immediately prior to surgery was 116.6 ± 42.3mm of Hg, which came down to 24.3 ± 14.5mm of Hg soon after surgery. Post operatively the gradient had increased to 38.8 ± 24.9mm of Hg over a follow up period of 4.3 ± 4 years. In our Group II patients the LVOT gradient at the time of diagnosis was 49.3 ± 39.3mm of Hg, and it had increased to 67.3 ± 44.7mm of Hg at the time of last follow up over a period of 7.8 ± 5.5 years. The post operative increase in LVOT gradients of 18.5 ± 18.5 mm of Hg in Group I patients in 4.3 ± 4 years, when compared with the net increase in LVOT gradients of 17.2 ± 24.7mm of Hg in 7.8 ± 5.5 years in Group II patients was not statistically significant [P value=0.44].

The number of patients with aortic regurgitation at the time of diagnosis and at the time of follow up were 17 and 19 patients respectively in operated group and

10 and 12 patients respectively in the nonoperated group respectively. The prevalence and progression of aortic regurgitation was not significantly different between the operated and nonoperated groups [Table 10].

Subgroup Analysis: Subaortic membrane-Aortic valve distance

Three of our patients had tunnel type of FSAS and these patients were excluded from this analysis {Table-9}. Of the remaining 35 patients, 13 patients who had subaortic membrane < 6mm of aortic valve formed the subgroup A. The sub aortic membrane was found at a distance of 3.1 ±1.5 mm from the aortic valve. The 22 patients who had subaortic membrane ≥ 6 mm from the aortic valve formed the subgroup B where the mean distance of the subaortic membrane from the aortic valve was 7.6± 2.1 mm. 10 patients in each subgroup had been operated and they formed theGroups I-A and I-B respectively.

Of the operated patients[Group I] the post operative increase in gradients during follow up were 27.8 ±18.7mm of Hg over 4 ± 3.9 years for subgroup A, and 8.2 ± 11.5 mm of Hg over 4.2 ± 4.2 years for subgroup B. This difference was statistically significant[P value=0.011]. Of the non operated patients [3 in subgroup A and 12 in subgroup B] the net increase in LVOT gradients were 23 ± 18.5 mm of Hg over 6.7 ± 5 years of follow up in subgroup A and 21.5 ± 26.6mm of Hg over 7.8 ± 6 years of follow up in subgroup B. This difference was not statistically significant.

Of the 13% patients with FSAS < 6mm from aortic valve, 12 had aortic regurgitation (92%) at the time of diagnosis and all the 13% had aortic regurgitation at last followup. Of the 22 patients with FSAS ≥ 6mm from aortic valve, 13 had aortic regurgitation (59%) at diagnosis and 18 (81%) at last followup [Table 10].

DISCUSSION

Our data confirmed that short segment narrowing is by far the most common form of FSAS. The 8% incidence of tunnel narrowing in our patients corresponds to what is reported in the literature. Sullivan et al had a 12% incidence of tunnel narrowing. Although strongly associated with congenital cardiac malformations, most evidence suggest that FSAS is an acquired lesion [31,32,37]. Other complex congenital malformations were excluded because their hemodynamic status and their influence on the natural history will be too different from the simple FSAS we had included in our study.

Pathogenesis

Many arguments have been proposed for the pathophysiologic concept of a primary hemodynamic abnormality of the LVOT, underlying the structural abnormality in FSAS [31,34]. Reduced aortic valve diameter and increased aortomitral seperation were identified in a pathological study. It was suggested that altered flow characteristic in a narrow and elongated left ventricular outflow tract during early heart development could cause embryonic cells near the crest of the ventricular septum to accumulate and later differentiate into ridge or band of tissue [38]. Recently Sigfussion et al showed that aortoseptal angle was steeper in patients with FSAS compared with general population [39]. However ours being a retrospective study we did not have enough data on all patients about aortoseptal angle or aortomitral discontinuity.

Recently Cape et al, [40] suggested a four stage etiology for the development of FSAS. They suggested that associated abnormalities in LVOT [stage 1] result in altered shear stress [stage 2], which triggers a preexisting genetic predisposition [stage 3], leading to proliferation of cells/structure in the LVOT [stage 4].

Associated lesions

We found clear association with congenital heart defects, 68.4% in our series. The youngest patient in our series was 3 years at the time of initial diagnosis. Sullivan et al found associated cardiac abnormalities in 71% of their patients[41]. VSD was the commonest associated lesion found in 23.7% of our patients. Most of patients [78%] had VSD above the FSAS [22%]. In a series of patients with FSAS and VSD reported by Freedom et al 73% of the VSDs were above the FSAS.[42]. Freedom et al found high association of RV and LV outflow obstructions [29%] occurring together in their series. Our incidence of this association was 13%. Baumstark et al reported a peculiar association of anomalous muscle bundles in RV creating a double chambered RV [43]. We had 2 cases [5%] of double chambered right ventricle in our series.

Sullivan et al noted persistence of the left superior venacava in 16% of their patients. They suggest that persistent left superior venacava and consequent dilated coronary sinus may restrict left ventricular filling during prenatal life and the diminished flow may contribute to the development of various downstream obstructions [41]. However we did not find this association in any of our patients.

Aortic regurgitation

The development of aortic regurgitation when FSAS is present is almost entirely the result both of tethering of the aortic valve and the trauma to the valve from the turbulent ejection jet during left ventricular systole [44]. The incidence of AR in our study was 71% at the time of diagnosis by doppler echocardiography and 67% by cardiac catheterisation. This incidence is very high compared with the 32% incidence at the time of diagnosis obtained by De'Vries et al [49]. However since more than 50% of their patients were diagnosed in the pre doppler echocardiography

era, their low incidence is likely to have been due to underestimation of aortic regurgitation. After a mean follow up period of 4.5 years they found that incidence of aortic regurgitation had increased to 81%. In our series after a mean follow up of 8.4 years new aortic regurgitation had developed in 4 patients [2 in operated and 2 in non operated groups] increasing the percentage of patients with aortic regurgitation from 71% to 81%.

Motro [45] et al found that the development of aortic regurgitation was correlated to the distance of the subaortic membrane to the base of the aortic valve cusps. We found a very high incidence of aortic regurgitation in our patients with subaortic membrane < 6mm from the aortic valve[92% at the time of diagnosis and 100% at last follow up] when compared with the subgroup with subaortic membrane ≥ 6mm from the aortic valve [59% at time of diagnosis and 81% at last follow up].

Balloon dilatation

Recently transluminal balloon dilatation of discrete subaortic membrane has been described as an useful complement to surgery [46,47,48]. However De Vries et al [49] do not advocate balloon dilatation because of the concern that FSAS being closely related to the mitral valve, dilatation could result in damage to the mitral valve apparatus. Balloon dilatation was attempted in three of our patients. The residual gradient was > 40 mm of Hg in all our patients. Rao et al strongly advocate balloon dilatation as an alternate first line of treatment of FSAS on the basis of their excellent immediate and intermediate term results[50] Similar results were obtained by Lebabid et al[51,52]. Rao suggests that patient selection is very important in deciding on balloon dilatation[53]. Patients with thickness of subaortic membrane less than 2 mm are most likely to be benefited. However the data regarding the thickness of the subaortic membrane was not available in our patients.

Surgery for FSAS

The mean age at the time of surgery was 18 years. The youngest patient operated was 3 years old, and the oldest 39 years. The mean LVOT gradient prior to surgery was 117 mm of Hg. No patient had a preoperative LVOT gradient < 60 mm of Hg. Compared to Brauner's series all our patients were older and had greater preoperative LVOT gradients. Brauner et al [54] series included patients with preoperative LVOT gradients < 40 mm of Hg also. They found that low pre operative gradients and younger age at surgery correlated with reduced recurrence of FSAS, and reduced risk of reoperations. However since all our patients had a higher preoperative gradient across the LVOT we could not analyze this variable in our series.

The mean postoperative LVOT gradient was 24 mm of Hg in our patients. 3 patients [13.6%] had a post operative gradients more than 30 mm of Hg. We had recurrent FSAS as defined by follow up gradients more than 50 mm of Hg in 5 patients [22.7%]. Of these 3 patients had FSAS closer to the aortic valve [<6mm] and 2 patients had FSAS beyond that. This difference however was not statistically significant. Our operative mortality was 5%[1out of 22 patients]. One patient died in early follow up due to infective endocarditis. Another patient died 6 years after resection of FSAS, during a surgery for aneurysm of valsalva. All the remaining patients are alive till the last follow up.

Predicting progression of FSAS

It was felt earlier that progression of FSAS was unpredictable. De'Vries et al in a retrospective study of 57 cases could not discover any constant morphological or hemodynamic features that could lead to a classification with satisfactory prognostic and predictive implications [49]. However they have not mentioned what

were the morphologic and hemodynamic parameters that they had looked for. Other investigators felt that different pathophysiologic mechanisms in FSAS cause specific forms that have different course and postoperative outcome[31,32,55]

Bezold et al, [56] in a recent study, devoloped an echocardiographic model for predicting progression of subaortic stenosis. They found that the FSAS-AV distance was 6.8+/- 3.8 mm in nonprogressive group and 3.5 +/- 2.4 mm in progressive group. We grouped our patients into two depending on the distance of FSAS from aortic valve was < 6 mm or ≥ 6 mm. We found that in our operated group of patients the post operative increase in LVOT gradients were significantly high if the FSAS was < 6mm from the aortic valve. 27.8 ±18.7 mm of Hg in 4 ± 3.9 years of followup in < 6mm subgroup and 8.2 ± 11.5 mm of Hg in 4.2 ± 4.2 years of followup in ≥ 6 mm subgroup [P value=0.011] .

However in our nonoperated group, though the net increase in LVOT gradients during the follow up period was marginally higher in the < 6 mm subgroup[23 ± 18.5 mm of Hg in 6.7 ± 5 years] when compared with the ≥ 6 mm subgroup[21.5± 26.6 mm of Hg in 7.8± 6 years], this difference was not statistically significant[P value=0.44].

The results of this analysis suggests that subaortic membranes close to the aortic valve tend to be more progressive than those away from the aortic valve. The other parameters suggested by Bezold [56]et al that can be used to predict progression include involvement of mitral valve by subaortic membrane and the pre-operative doppler gradient.

CONCLUSIONS

1. Discrete short segment narrowing is the commonest form of fixed subaortic stenosis and tunnel type narrowing is less common.

2. Fixed subaortic stenosis is commonly associated with other congenital heart defects.

3. Aortic regurgitation is commonly assocaited with fixed subaortic stenosis-closer is the narrowing to aortic valve greater is the prevalence of aortic regurgitation.

4. Fixed subaortic stenosis is a progressive disorder and left ventricular outflow tract gradient continues to progress even after surgical correction, and hence these patients need close follow up after surgery.

5. Distance of the subaortic narrowing from the aortic valve can be used to predict the progression of left ventricular outflow tract obstruction, particularly after surgery-closer the narrowing to the aortic valve greater is the progression.

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Table1
BASELINE CHARACTERISTICS

	No.	%
No. of Patients	38	100
Females	16	42
Males	22	58
Age (Range)	**Average**	**Std.Deviation**
Diagnosis (1 to 34)	12	8
Cath (3 to 36)	15.6	8.5
Surgery (3 to 39)	18.3	8.8

Table 2
CLINICAL FEATURES

	No.	%
Asymptomatic	4	10.5
Syncope	2	5.3
Angina	8	21
Dyspnea	32	84
Rheumatic Fever	1	2.6
Infective.Endocarditis	2	5.3
Congestive Heart Failure	2	5.3
Ejection Click	3	7.9
Reversed Split A2	2	5.3
LVH	36	94.7
LVH with Strain	18	47.4
Cardiomegaly	8	21
Ascending Aortic Diltation	10	26.3
Tunnel Narrowing	3	7.9
Discrete Narrowing	35	92.1

Table 3
CATHETERISATION DATA

	No.	%
No AR	12	31.6
Mild AR	21	55.4
Moderate AR	5	13.2
Severe AR	0	0
	Average	**Std.Deviation**
LVOT Gradient in mm of Hg	88.5	52.9
FSAS-AV Distance in mm	6.8	2.8

Table 4
ASSOCIATED ANOMALIES

	No.	%
Total	26	68.4
VSD	9	23.7
PDA	8	21.1
Mitral Regurgitation	5	13.2
Pulmonary Hypertension	4	10.5
Bicuspid Aortic Valve	3	7.9
Mitral Stenosis	3	7.9
Supramitral Ring	2	5.3
Coarctation of Aorta	3	7.9
Infundibular PS	2	5.3
DCRV	2	5.3
Valvar PS	1	2.6
Single Coronary Artery	1	2.6
Coronary Artery Aneurysm	1	2.6
Sinus of Valsalva Aneurysm	1	2.6
Aorto Pulmonary Window	1	2.6
Cleft AML	1	2.6
Cleft PML	1	2.6
Single Papillary Muscle	1	1 2.6
IVC Interruption	1	2.6
Membranous Septal Aneurysm	1	2.6
Rubella Syndrome	1	2.6
LV Dysfunction	1	2.6
ASD	1	2.6

Table 5
SURGICAL PROCEDURES DONE

	No.	%
Total No.of Patients	38	100
No.Operated	28	73.6
No.Operated for FSAS	22	57.9
Operated, but not for FSAS	6	15.8
Not Operated	10	26.3
Balloon Dilation Done	3	7.9

Table 6
SURGICAL PROCEDURES DONE

SURGERY DONE OPERATED GROUP	No.
FSAS Excision	22
MVR	3
AVR	2
Balloon Dilatation of FSAS	3
Balloon Dilatation of Coarctation of Aorta	1
Coarctation Repair	2
PDA Ligation	4
VSD Closure	4
ASD Closure	1
Aortic False Aneurysm Repair	1
Kay's annuloplasty of Mitral	1
Cleft PML/AML Repair	1
Myomectomy/Myotomy	2
Infundibular Resection	2
Aortic Valve Commisurotomy	1
SURGERY DONE : NON OPERATED GROUP	No.
PDA Ligation	3
VSD Closure	2
Coarctation Repair	1

Table7
AR IN OPERATED/NONOPERATED GROUPS

	Operated=22		Non operated=16	
AR	Diagnosis	Follow up	Diagnosis	Followup
NIL	5	3	6	4
MILD	15	9	8	4
MOD	2	9	1	6
SEV	0	1	1	2
NET AR	17	19	10	12

Table 8a
LVOTO SUBGROUP ANALYSIS : OPERATED GROUP

Operated for FSAS, n=22	Mean	Std. Deviation
LVOTO at Diagnosis (mm of Hg)	81.4	37.8
LVOTO Pre Op.(mm of Hg)	117	42.3
LVOTO Post Op. (mm of Hg)	24.3	14.5
LVOTO Followup (mm of Hg)	38.8	24.9
P.Op.Gr> 30mm	3 patients	13.6%
F.Up.Gr > 50mm	5 patients	22.7%
Pre Op.Increase in LVOTO (mm of Hg)	32.6	14.7
Pre Op.Follow Up (YRS)	5.3	4.8
Post Op.Increase in LVOTO (mm of Hg)	18.5	18.5
Post op. Followup (yrs)	5.3	4.8

Table 8b
LVOTO SUBGROUP ANALYSIS : NON-OPERATED GROUP

Not Operated for FSAS , n=16	Mean	Std. Deviation
LVOTO at Diagonis (mm of Hg)	49.3	39.3
LVOTO Follow up (mm of Hg)	67.3	44.8
Net increase in LVOTO (mm of Hg)	17.2	24.7
Net followup period (years)	7.8	5.5

Table 9
SUBGROUPS ANALYSIS
FSAS- AORTIC VALVE DISTANCE (LVOTO)

NOT OPERATED FOR FSAS (n=15)		
Variable	SAM <6mm (n=3)	SAM ≥ 6mm (n=12)
LVOTO Cath (mm of Hg)	60 ±20	45.6 ±36.5
LVOTO Diag (mm of Hg)	59.3± 20	39.4 ±35.9
LVOTO F.Up (mm of Hg)	82.3 ±19.5	53.6± 33.2
LVOTO Increase (mm of Hg)	23 ±18.5	21.5 ±26.6
Followup.Duration (yrs)	6.7± 5	7.8± 6
OPERATED FOR FSAS (n=20)		
Variable	SAM< 6mm (n=10)	SAM ≥ 6mm (n=10)
LVOTO Cath (mm of Hg)	99.5 ±56.6	129 ±36
LVOTO Diagnosis (mm of Hg)	57.4 ±16.4	98.7 ±50
LVOTO Pre.Op (mm of Hg)	105.6 ±48.3	127.2 ±35
LVOTO Po.Op (mm of Hg)	24.9 ±20	22.5 ±9.2
LVOTO Followup (mm of Hg)	44.3 ±23.1	33.3 ±25.2
LVOTO Incre.Pre.Op. (mm of Hg)	27.±5 25.9	32.2 ±16.7
Pre Op. Follow up Dur. (yrs)	4.7 ± 4	5.5 ± 5.6
LVOTO Incr Po.Op. (mm of Hg)*	27.8 ±18.7	8.2 ± 11.5
Po.Op.F.Up.Duration (yrs)	4 ±3.9	4.2 ± 4.2

*P Value 0.011 for others P = NS

Table 10
SUBGROUPS ANALYSIS
FSAS- AORTIC VALVE DISTANCE (AR)

	FSAS-AV Dist < 6mm (n=13)		FSAS-AV Dist ≥ 6mm (n=22)	
AR	**Diagnosis**	**Followup**	**Diagnosis**	**Followup**
Nil	0	0	9	4
Mild	10	5	12	10
Mod.	1	4	1	8
Sev.	1	1	0	0
Net	12 (92%)	13 (100%)	13 (59%)	18 (81%)