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#### Dear Friends,

Percutaneous Transluminal myocardial septal ablation (PTSMA) has emerged as a less invasive treatment of symptomatic patients with hypertrophic obstructive cardiomyopathy (HOCM). In the past three decade, the availability of this sophisticated technique has revived the interest of cardiologists in left ventricular outflow tract obstruction, which has led to the recognition that most patients with hypertrophic cardiomyopathy (HCM) have the obstructive type. Follow-up studies have already shown the safety and efficacy of the procedure, which offers symptomatic relief in most patients. Long-term survival is comparable to historical reports after surgical myectomy. Complications are rare and can be further reduced by an increase in the experience of the operators, while the theoretical concern for possible ventricular arrhythmogenicity caused by the myocardial scar has not been documented by the existing data. Although there are still no randomised trials, percutaneous septal ablation is a viable alternative for patients with HOCM. We have one of the largest series of such therapy in India (Table-4).

- Dr Milan Chag

# HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY: ALCOHOL SEPTAL ABLATION- A PROVEN THERAPY NOW!

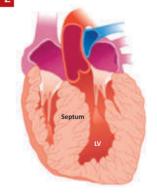
#### INTRODUCTION AND SALIENT FEATURES:

Hypertrophic cardiomyopathy (HCM) is a primary myocardial disorder which is clinically defined by the presence of unexplained left ventricular hypertrophy (Figure-1 & 2).

#### Figure-1



Figure-2



- The most common genetic cardiac disease, affecting 1 in 500 individuals.
- It is inherited as an autosomal dominant trait with variable penetrance, most commonly involving sarcomeric protein mutations.
- The disease can be diagnosed in patients of all ages and presents as asymptomatic individual to patients with severe symptoms of exertional dyspnoea or angina and reduced exercise capacity.
- The natural history of the disease may be highly heterogeneous with life expectancy ranging from normal longevity to sudden arrhythmic death (Table 1), often presenting at a young age, or evolution to congestive heart failure atrial fibrillation or stroke.
- Most patients present a characteristic left ventricular morphology with hypertrophy of the basal interventricular septum that is coupled with systolic anterior motion (SAM) of the anterior mitral valve leaflet and leads to

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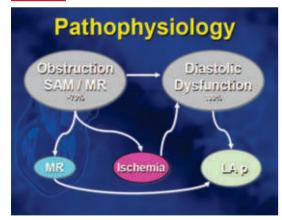
### Table-1 Risk Factors For **Sudden Cardiac Death**

- 1. Familial sudden death
- 2. Unexplained syncope
- 3. Multiple, repetitive NSVT (Holter)
- 4. Abnormal exercise response
- 5. Massive LVH Septal thickness > 30 mm
- 6. Cardiac arrest survivors
- 7. Documented sustained ventricular tachycardia
- 8. Cardiac MRI: >15% area with LGE

dynamic left ventricular outflow tract (LVOT) obstruction and mitral regurgitation due to malcoaptation of the mitral leaflets.

Symptomatic status depends on left ventricular obstruction, diastolic dysfunction and myocardial ischaemia. The existence of significant obstruction at rest or after provocation is associated with symptomatic status and has significant prognostic implications. (Figure-3)

#### Figure-3



#### **MANAGEMENT:**

In general, treatment of patients with hypertrophic cardiomyopathy aims at relieving symptoms, reducing the risk of sudden death and offering genetic counselling.

- Consequently, treatment of symptomatic patients with obstructive HCM (HOCM) aims at the reduction of the pressure gradient. Medical treatment with ß-blockers, disopyramide or verapamil, are first line therapy but some patients remain Figure-4 symptomatic in spite of them. Mavacamten (cardiac myosin inhibitor) is a novel new agent. It has shown significant improvement in symptoms, hemodynamics and cardiac remodeling on short term follow up, Reduction in LVFE due to it needs regular monitoring.
- In drug-refractory patients, alcohol septal ablation (Percutaneous Transluminal Septal Myocardial Ablation-PTSMA) (Figure-4)

has emerged as a less invasive treatment than surgery to reduce LVOT obstruction by creating an infarction limited to the part of the basal septum, involved in the development of LV obstruction (Table-2).



#### PROCEDURE (PTSMA)

Under local anesthesia and prophylactic transjugular temporary pacemaker lead insertion, first septal artery is identified and OTW balloon

#### Table-2: INDICATIONS FOR SEPTAL REDUCTION (PTSMA) TREATMENT:

#### **CLINICAL INDICATION**

- Symptomatic patients
  - ☐ Drug-refractory or severe side effects of drugs
  - ☐ Functional class III and IV or functional class II with objective exercise limitations
  - ☐ Recurrent exercise-induced syncope
- Failure of prior myectomy or pacemaker
- Comorbidity-related increased surgical risk

#### **HAEMODYNAMIC INDICATION**

- Intracavitary gradient >30 mmHg at rest and/or
- Provocable gradient >50 mmHg

#### **MORPHOLOGIC INDICATION**

- Echocardiography
  - ☐ Subaortic, HCM with SAM-associated gradient
  - Exclusion of intrinsic mitral valve apparatus disorders
- Coronary angiography
  - Suitable septal branch





of appropriate size is placed over the Figure-6: Procedure (PTSMA) guide wire in the artery to occlude it completely. After confirming the target septal tissue by contrast echocardiography, 1 to 3 ml of absolute alcohol (1 ml/1 cm of IVS) is injected in target septal artery through central lumen of OTW balloon catheter under continuous ECG and hemodynamic monitoring. At the end, balloon catheter is removed, check angiogram is done and patient is monitored in CCU for 48 hours. (Figure-5, 6)

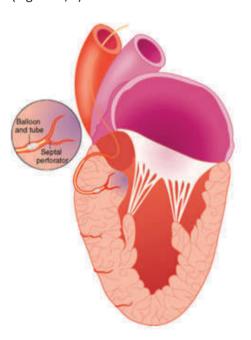
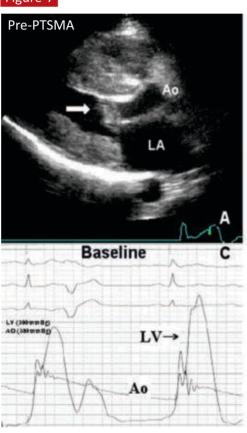




Figure-7



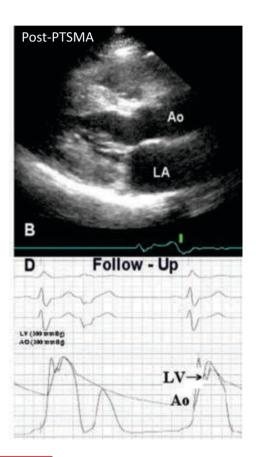
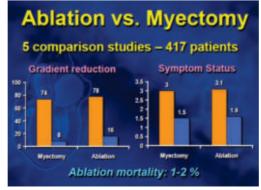


Figure-5 : Procedure (PTSMA)

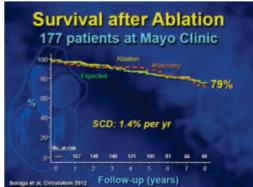
#### PATHOPHYSIOLOGICAL EFFECTS OF SEPTAL **ABLATION:**

- Injection of alcohol during the procedure causes coagulative necrosis of the targeted myocardium and the septal micro vasculature.
- Tissue oedema appears early in this process, while muscle replacement

Figure-8



#### Figure-9







by scar formation develops only after several days.

 Thinning of the ablated area and scar formation lead to a permanent and significant reduction of the obstruction and the associated mitral regurgitation within the next 6-12 weeks.

#### **CLINICAL RESULTS**

- Haemodynamic success with reduction in both resting and provocable gradients is accomplished in ≥ 90% of patients and is associated with significant improvement in symptoms (Figure-7,8).
- Mean NYHA class decreased from 2.9 to 1.2 and mean CCS class decreased from 1.9 to 0.4 at 1-year follow-up. Exercise capacity also improved on a treadmill. In a cohort of the first 99 consecutive patients treated the overall survival was 98% at 12 years, while 80% of patients

Table-3: Comparison of Septal Myectomy and Percutaneous Alcohol Septal Ablation					
Parameter	Percutaneous Alcohol Septal Ablation	Surgical Myectomy			
Invasiveness	Percutaneous access	Sternotomy			
Onset of reduction in LVOT	Maximam decrease in gradient instantly,				
Instantaneous					
gradient	And by 6 to 12 week				
Success rate (%)	>90%	>90%			
Procedural mortality (%)	<1%	2-10%			
Recovery time	2-4 days	2 week			
Effect on LVOT gradient	Decreases to <20 mm Hg	Decreases to			
		<10 mm Hg			
Postprocedure conduction	Right bundle branch block	Left bundle branch			
abnormality (commonenst)		block			
Need for permanent	5%	3-10%			
pacemaker-all patients (%)					
Length of follow-up (year)	15-20	30			

remained free of severe symptoms, atrial fibrillation, and stroke or ICD implantation. Results of last 12 years are comparable to published literature (Table-3, Figure-9)

### **CONCLUSIONS**

Alcohol septal ablation has emerged in the last 20 years as a less invasive alternative to the standard surgical treatment of symptomatic patients with HOCM. The accumulated long-term results have shown an ongoing relief of symptoms in the majority of patients. Hospital mortality can be practically eliminated in experienced centers, while the need for permanent pacing has also been reduced with increased experience.

Table-4: Results										
STUDY	NO. OF	AGE	PACEMAKER	MEN	IN-HOSPITAL	LONG-TERM	REDO	МҮЕСТОМҮ	SURVIVAL	SURVIVAL
	PATIENTS	(YEARS)	IN-HOSPITAL	FOLLOW-UP	MORTALITY	ALL-CAUSE	PROCEDURES	(%)		WITHOUT
			(%)	TIME (YEARS)	(%)	MORTALITY (%)	(%)			SYMPTOMS
Seggewiss 2007	100	52.7±15.7	8	4.8±1.2	1	3			96%@8y	74 %
Welge 2008	347	54±15	7	4.8±2.9	1	8	5	3	92%	74 %
Sorajja 2008	138	64±21	20	2.2±2.8	1.4	8	4		88%@4y	76.4 %
Kuhn 2008	329	58±15	17	2.1	1.8	7	13		91.1%	
Fernandes 2008	619	53.9±15.0	8.2	4.6±2.5	1	8	14	25	89%@8y	
Kwon 2008	55	63±13	25.5	8±1	0	24	5.5	5	76%@10y	
Noseworthy 2009	89			5.0±2.3		9	10	11	91%	
Ten Cate 2010	91	54±15	4	5.4±2.5	2	10	5	5	88%	
Lyne 2010	12	69±22	0	11.75	0	3	17	0		73%@10y
Chag MC 2023	99	46±12	3	12.4	0	2	2	0	98 %	80 %







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