Outcomes and Complications of alcohol septal ablation for younger patients with hypertrophic obstructive cardiomyopathy: a single-centre study

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Backgrounds: Hypertrophic cardiomyopathy (HCM) is a genetic disorder of cardiac muscle with a heterogeneous clinical course. Alcohol septal ablation is a treatment option for severely symptomatic drug-refractory hypertrophic obstructive cardiomyopathy. This study aimed to determine the outcome of alcohol septal ablation in 20 patients from a single center in Iraq.
Methods: Twenty patients with age less than 60 years had undergone alcohol septal ablation between May 2013 and February 2019 at Nasiriya Heart Centre. Patients were selected for alcohol septal ablation depending on the clinical and angiographic suitability of septal perforator branches. Clinical, electrocardiographic, and echocardiographic parameters were evaluated in the periprocedural period and during follow-up.
Results: Only three patients (15 %) remained in NYHA class III after 6 months of follow-up, one of them underwent repeated alcohol septal ablation with successful improvement at 6 months follow-up, and the other two patients awaiting reevaluation at 6 months to decide for repeating ASA. Significant reduction of left ventricle outflow gradients (LVOTG) and septal thickness were observed during 6 months follow-up. Beyond 6 months, except for 3 patients, there was no further decrease in either septal thickness or LVOTG noted. The incidence of right bundle branch block (RBBB) after ASA was 45 % % and 3 patients (15 %) needed PPM implantation. There was no cardiovascular death on follow-up.
Conclusion: Alcohol septal ablation is a safe and effective option for severely symptomatic patients, less than 60m years with HOCM because of its low risk and its significant clinical, echocardiographic, and hemodynamic improvement. The overall in-hospital adverse cardiovascular events were low and few patients required in-hospital permanent pacemaker implantation.

Key words: hypertrophic cardiomyopathy, alcohol septal ablation, sudden death, hypertrophic obstructive cardiomyopathy, left ventricular hypertrophy.

Výsledky a komplikace alkoholové septální ablace u mladších pacientů s hypertrofickou obstrukční kardiomyopatií: studie prováděná v jednom centru

Úvod: Hypertrofická kardiomyopatie je dědičné onemocnění srdečního svalu, které se vyznačuje různorodým klinickým průběhem. Alkoholová septální ablace (ASA) je jednou z možností léčby v případě vysoce symptomatických pacientů s hypertrofickou obstrukční kardiomyopatií, která je refrakterní k farmakoterapii. Cílem této studie bylo stanovit výsledek alkoholové septální ablace u 20 pacientů z jednoho centra v Iráku.
Metody: V období od května 2013 do února 2019 podstoupilo 20 pacientů mladších 60 let alkoholovou septální ablaci v Nasiriya Heart Centre v Iráku. Pacienti byli indikováni k alkoholové septální ablaci na základě klinické a angiografické vhodnosti septálních

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větví. V periprocedurálním období a v průběhu sledování byly hodnoceny klinické, elektrokardiografické a echokardiografické parametry.

Výsledky: Po šesti měsících sledování zůstali pouze tři pacienti (15 %) v třídě NYHA III, přičemž jeden z nich podstoupil opakovanou alkoholovou septální ablaci se zlepšením v šesti měsících sledování a zbylí dva pacienti čekali na přehodnocení v šesti měsících, aby se rozhodlo o opakování ASA. Během šesti měsíců sledování jsme pozorovali významné snížení tlakového gradientu ve výtokovém traktu levé komory (LVOT) a tloušťky septa. Po uplynutí šesti měsíců jsme, s výjimkou tří pacientů, žádné další snížení tloušťky septa ani LVOT nezaznamenali. Incidence blokády pravého Tawarova raménka po ASA byla 45 % a u tří pacientů (15 %) byla nutná implantace permanentního kardiostimulátoru. Během období sledování nedošlo k žádnému úmrtí z kardiovaskulárních příčin.

Závěr: V případě vysoce symptomatických pacientů s hypertrofickou obstrukční kardiomyopatií ve věku pod 60 let je alkoholová septální ablace bezpečná a účinná metoda, a to pro své nízké riziko a významné klinické, echokardiografické a hemodynamické zlepšení. Celkový výskyt nemocničních nežádoucích kardiovaskulárních příhod byl nízký a jen u mála pacientů byla nutná implantace permanentního kardiostimulátoru.

Klíčová slova: hypertrofická kardiomyopatie, alkoholová septální ablace, náhlé úmrtí, hypertrofická obstrukční kardiomyopatie, hypertrofie levé komory.

Introduction

Hypertrophic cardiomyopathy (HCM) is a genetic disorder of cardiac muscle with a heterogeneous clinical course (1). In up to 60%, the disease is an autosomal dominant trait caused by a mutation in cardiac sarcomere protein genes (2–6). About five to ten percent of adult cases are caused by other genetic disorders (7, 8). Some patients have non-genetic disorders that mimic genetic forms of the disease, for example, senile and AL amyloidosis (9, 10). HCM affects 1 out of 500 individuals in the general population (11) and maybe more prevalent in men than women, although the gender difference in prevalence has not been confirmed (12).

The disease is clinically characterized by left ventricular hypertrophy (LVH) which is typically asymmetric and not solely explained by abnormal loading conditions (12). A subgroup of HCM patients has left ventricle outflow tract obstruction (LVOTO) caused by systolic anterior motion (SAM) of the anterior mitral valve leaflet (AMVL) (13, 14). Approximately one-third of patients with HCM have a physiological pattern of obstruction at rest worsened with exertion, one-third solely during exertion, and one-third have non-obstructive HCM (14).

At rest, LVOTO is associated with limiting symptoms (dyspnea, angina, syncope) and a worse prognosis (15–19). Variable phenotypic penetrance and symptoms may sometimes result in the diagnosis being established when affected individuals reach adulthood with severe myocardial dysfunction at presentation (20–22). An instantaneous Doppler LVOTG of ≥ 30 mmHg is considered significant, and such patients are classified as having the obstructive form of the disease. HOCM is considered to be hemodynamically significant only when the LVOTG is ≥ 50 mmHg. [14] Latent LVOTO can be demonstrated by exercise, Valsalva maneuver, postural changes or an isoprenaline infusion (17, 23–25).

Currently, there is no evidence that asymptomatic patients with LVOTO benefit from treatment to reduce the severity of obstruction; treatment is reserved for patients with LVOTO and drug-refractory symptoms (12, 26–29).

Pharmacological therapy consists of non-vasodilator beta-blockers (BB), calcium channel blockers (CCB), and disopyramide which modulates the dynamic physiology of obstruction through their negative inotropic and chronotropic effects (30–35).

In the 5% to 10% of patients who remain highly symptomatic despite optimal medical therapy (OMT) or unacceptable side effects, septal reduction therapy is indicated, either by surgical myectomy or alcohol septal ablation (ASA) following a comprehensive evaluation of the mechanism of obstruction (12, 26, 36–39). Factors in favor of ASA are advanced age, presence of comorbidities increasing the risks of cardiac surgery, history of cardiac surgery, failed previous surgical myomectomy, and patients with a right bundle branch block (RBBB), given the high risk of left bundle branch block (LBBB) induced by surgical myomectomy (40–45).

Surgical myectomy is a technically demanding operation, but with improved surgical techniques and perioperative care, the current perioperative mortality in high volume centers is low (46–48). The choice of therapy should be based on a detailed assessment of septal anatomy and mitral valve (MV) (49–56).

The aim of this study is to describe the long-term outcomes of ASA procedures for younger patients with HOCM.

Patients and Methods

We performed a prospective single-center study of HOCM patients who underwent ASA between May 2013 and February 2019 at Nasiriya Heart Centre, Iraq. A total of 34 patients with symptomatic HOCM refractory to OMT were referred to our center for consideration of ASA. All patients were suitable for both ASA and myectomy. The reason for the selection of ASA over surgical myectomy was the patient's preference for percutaneous intervention rather than open-heart surgery.

Initially, twelve patients were excluded from the study with three patients had their symptoms improved after more optimization of medical therapy. One patient was 16 years old age. One patient had a mid-cavity LV obstruction and seven patients were unsuitable due to very small septal artery; they were sent for surgical myectomy.

Twenty-two patients proceeded to ASA. Two other patients were excluded because their procedures were abandoned before alcohol injection: one patient developed pericardial tamponade due to right ventricular perforation by temporary pacemaker lead and the in the other patient, the balloon

could not be delivered into the target septal branch. Subsequently, our study enrolled twenty patients who underwent complete ASA procedures. The enrollment scheme is shown in Figure 1.

Methods

All patients presenting with criteria for septal reduction therapy were evaluated at Nasiriya Heart Centre. All the following criteria together are mandatory for inclusion: Adult patients (more than 18 years old age) with HCM (defined by a wall thickness ≥ 15 mm in one or more LV myocardial segments that is not explained solely by loading conditions), LVOT obstruction caused by SAM of AMVL with LVOTG ≥ 30 mm Hg at rest or ≥ 50 mmHg with provocation, persistent severe symptoms despite OMT including correction of exacerbating factors.

HOCM-related severe cardiovascular symptoms were defined as New York Heart Association (NYHA) functional class III/IV dyspnea, Canadian Cardiovascular Society (CCS) class III/IV angina, and/or exertional pre-syncope/syncope without alternate explanations. NYHA and CCS functional classes were defined as mentioned in the original source (86, 87) other requirements for inclusion are life expectancy > 1 year and informed patient's consent.

The exclusion criteria are basal septal wall thickness (at the point of mitral-septal contact) < 17 mm, moderate to severe primary MR (not by SAM), the need for concomitant cardiac surgical procedure (e. g., bypass grafting, valve replacement), mid-cavity obstruction, absence of suitable septal branch, technically difficult septal branch, procedural complications that mandate the procedure to be postponed before alcohol injection into target septal branch and presence of comorbidities that would compromise clinical improvement.

The diagnosis of HCM was based on typical clinical, electrocardiography (ECG), and echocardiographic features, with LVH occurring in the absence of any other cardiac or systemic disease that could have been responsible for the hypertrophy.

All patients were evaluated for the following characteristics: age, sex, HOCM-related symptoms (NYHA class, CCS class, and exertional presyncope/syncope), drugs (types, doses, and side effects), and conventional risk factors for SCD, comorbidities, AF and baseline ECG for conduction abnormalities.

All eligible patients underwent coronary angiography using transfemoral artery approach to assess for the presence of myocardial bridging, congenital coronary artery anomalies, or a significant atherosclerotic coronary artery disease and to delineate the anatomy, position, and suitability of left anterior descending (LAD) septal perforator branches.

The procedures of alcohol ablation were carried out under conscious sedation and the most proximal and sizable septal branch is usually selected. A 0.014″ coronary wire is inserted into the selected septal branch. An over-the-wire balloon of 1.5–2.5 mm diameter is inflated within the artery. The balloon's lumen allows selective delivery of angiographic contrast, echo contrast, and ultimately, alcohol into the selected septal artery.

Echocardiography was used for guidance. Echo contrast confirmed that the myocardial volume subtended by the selected septal artery is an appropriate target for ablation. The appropriate target lies adjacent to the point of mitral-septal contact. If the contrast agent did not localize exclusively to the basal septum at and adjacent to the point of mitral-septal contact, the procedure was abandoned.

A temporary pacing wire was inserted through the femoral vein (except in patients who already had PPM or ICD) prior to the injection of alcohol in case significant bradyarrhythmias follow conduction system damage.

Approximately 0.1 ml of ethanol (concentration > 95 %) per 1 mm of a thickness of the target myocardium is injected slowly (1 ml/minute). The balloon remains inflated for 5–10 minutes post-ethanol injection to prevent reflux in the LAD and to enhance delivery at the target myocardium.

Immediate procedural success is defined by at least 50 % reduction in LVOTG by invasive hemodynamics at rest or, among those with predominantly labile obstruction, after provocation with a final residual gradient of 20 mmHg or less in the absence of death or need for emergency surgery. In addition, the infarcted septal zone appears echo-bright, and ECG may reveal a new RBBB and ST-segment elevation in leads V1-V3 with reciprocal changes in the lateral leads.

Following completion of the procedure, patients were admitted to coronary care. All patients underwent TTE daily for assessment of LVOTG and to exclude procedure-related complications. Procedural failure is defined as the persistence of both symptoms and LVOTG (resting or provoked).

After discharge, patients were followed-up for thirty days and long term adverse events including new conduction disturbance, adverse arrhythmic events (AAE) (sudden cardiac death (SCD), resuscitated cardiac arrest due to ventricular fibrillation (VF) or ventricular tachycardia (VT), and appropriate ICD shock), need for PPM or ICD implantations, infective endocarditis, cerebrovascular accident (CVA) and HCM-related death (death due to heart failure, CVA or SCD).

In addition, clinical and TTE follow-up at set periods; 4 weeks, 6 months, 12 months, and annually thereafter was done for NYHA class, CCS class, patients' medications, septal wall thickness, LVOTG (resting and provoked), and the need for additional septal reduction therapy (e.g. surgical myectomy, repeat ASA).

Successful ASA at 6 months follow-up is defined as successful symptomatic relief (NYHA class ≤ 2 and/or CCS class ≤ 1) with an improvement of at least 1 class combined with a resting LVOTG < 30 mmHg and a provoked LVOTG < 50 mmHg. Patients in whom LVOTO is successfully abolished are still subject to other HCM-related risks and outcomes. Patients had been evaluated to address family screening, risk of SCD, atrial arrhythmias, and prevention of CVA.

Statistical analysis

The statistical analysis included descriptive data using number and percentages. The p value of significant associations was measured using chi square and t test. The data were processed and analyzed by using computer software SPSS (Statistical package for social science) version 22.

Results

Patient Characteristics

Baseline characteristics of the study group are shown in Table (1). Of the study group, 13 patients were males (65 %), 13 patients

Tab. 1. *Baseline characteristics*

Age in years (mean ± SD)				36.6 ± 10
Age group No (%)	20–39 40–60			13 (65) 7 (35)
Males No. (%)				13(65)
Symptoms No (%)	Angina Dyspnea Syncope/pre-syncope Palpitation			8 (40) 20 (100) 0 0
AF No (%)				0
VT/VF No. (%)				0
Medications No (%)	BB CCB Disopyramide			20 (100) 8 (40) 0
Family history of	HCM	No (%)		5 (25)
Prior	ICD PPM	No (%) No (%)		2 (10) 0
Reason for selection of ASA over surgery	Patient preference No. (%) Other			20 (100) 0
Mean Septal wall thickness in mm (mean ± SD)				30 ± 4.4
Extreme septal hypertrophy (≥ 30 mm) No. (%)				9 (45)
LVOTG mean in mmHg (mean ± SD) resting provoked				44 ± 28 87.7 ± 42
MR No (%)	Trivial-mild Moderate Severe			12 (60) 8 (40) 0
EF < 50 % No (%)				0
Diastolic function	Normal Grade 1 Grade 2 Grade 3, 4	 dysfunction dysfunction dysfunction	 No (%) No (%) No (%)	 16 (80) 4 (20) 0
LA diameter in mm (mean ± SD)				3.8 ± 0.29
ECG conduction disturbance No. (%)				1 (5)
Prior	ASA MYECTOMY			0 0

AF – atrial fibrillation; VT – ventricular tachycardia; VF – ventricular fibrillation; BB = beta blockers; CCB – calcium channel blockers; HCM – hypertrophic cardiomyopathy; SCD = sudden cardiac death; ICD – implantable cardiac defibrillator; PPM – permanent pacemaker; ASA – alcohol septal ablation; mm – millimeter; LVOTG – left ventricular outflow tract gradient; MR – mitral regurgitation; EF – ejection fraction; LA – left atrium; ECG – electrocardiogram

Tab. 2. *Procedural parameters and results*

The volume of alcohol in cc (mean ± SD)		2.95 ± 0.42
Use of contrast TTE (%)		100
Procedure time in minutes (mean ± SD)		56 ± 13
Septal arteries injected per procedure (no.)		1
Maximum LVOTG in mmHg (mean ± SD)		21.5 ± 23
ECG septal Q		2 (10)
Complications No. (%)	Anterior MI	0
	RBBB	9 (45)
	CHB	3 (15)
	VT/VF requiring treatment	0
	Access site bleeding	0
	Pericardial tamponade	0
	CVA	0
	VSR	0
	Death	0

TTE – transthoracic echocardiography; LVOTG – left ventricular outflow tract gradient; ECG – electrocardiogram; MI – myocardial infarction; RBBB – right bundle branch block; CHB – complete heart block; VT – ventricular tachycardia; VF – ventricular fibrillation; CVA – cerebrovascular accident; VSR – ventricular septal rapture.

(65%) were young adults (20–39 years) and 7 patients (35 %) were middle-aged adults (40–60 years). All patients had exertional dyspnea with NYHA class 3 and 8 of them (40 %) had angina with CCS class (1–3). None had previous ASA, myectomy, or cardiac surgery. Two patients had a prior implanted ICD, one for unexplained syncope and the other for asymptomatic non-sustained VT. None of the patients had AF. All patients were on beta-blockers, 8 patients (40%) were receiving verapamil, and none was on disopyramide.

Procedural complications

Three patients (15 %) eventually developed CHB requiring a permanent pacemaker, none of them had baseline ECG conduction abnormalities, two of them had transient intraprocedural CHB and postprocedural RBBB and one patient had persistent intra and postprocedural CHB. In patients not requiring a PPM, the most common new conduction abnormalities were RBBB in 9 patients (45 %), while one patient developed transient intraprocedural CHB that did not recur later on. The development of intraprocedural CHB was the only factor significantly associated with the risk of PPM implantation. There was no alcohol reflux injury to LAD and no ventricular septal rapture (VSR). There were no periprocedural AAE, death, or stroke. Procedural results are shown in table (2) and periprocedural parameters of patients' required PPM are shown in table (3).

Treatment results

Figures (2 and 3) and Table (4) show the clinical and echocardiographic outcomes of ASA procedures.

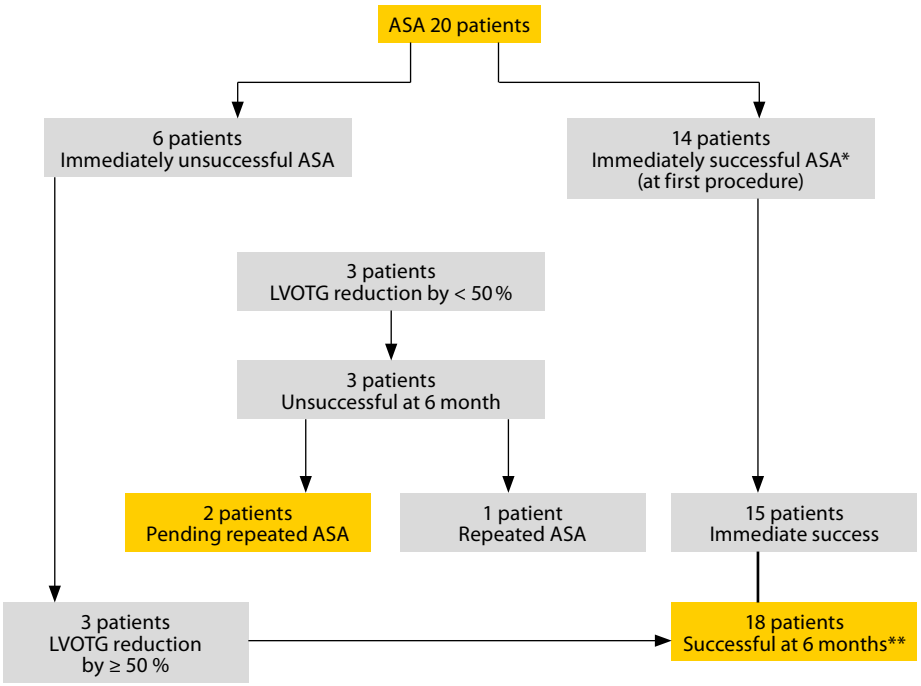
Immediate successful reduction in LVOTG occurred in 15 patients (75 %). Those patients also had a reduction in resting and provoked LVOTG at all-time points (5 days and 6 months) and a reduction in septal thickness at 6 months, indicative of progressive remodeling. The symptoms improved at 6 months

Tab. 3. *Periprocedural parameters of patients requiring PPM after ASA*

Periprocedural parameters	Required PPM	Did not require PPM	P value
No. (%)	3 (15)	17 (85)	
Age in years (mean ± SD)	34.6 ± 9	36.8 ± 10	0.74
Baseline LVOTG (mmHg) Resting (mean ± SD) Provoked (mean ± SD)	 30 ± 0 60 ± 10	 47 ± 29 93 ± 45	 0.35 0.05
Ethanol dose ≥ 3CC	1 (33.3)	9 (52.9)	0.53
Baseline conduction disturbance No. (%)	0 (0)	1 (5.9)	0.66
Intraprocedural CHB No. (%)	3 (100)	1 (5.9)	< 0.005
Post procedural RBBB No. (%)	2 (66.7)	7 (41.2)	0.42

LVOTG – left ventricular outflow tract gradient; CHB – complete heart block; RBBB – right bundle branch block

Fig. 2. *The outcome of ASA procedures immediately and at 6 months*



** Immediate success is defined as the reduction in LVOTG by more than 50 % and to less than 20 mmHg.*
*** Success at 6 months is defined as successful symptomatic relief (≤ NYHA class 2 and/or ≤ CCS class 1) with an improvement of at least 1 class combined with a resting LVOTG < 30 mmHg and a provoked LVOTG < 50 mmHg.*
ASA – alcohol septal ablation; LVOTG – left ventricular outflow tract gradient

Tab. 4. *Change in clinical and echocardiographic parameters at 6 months*

	Mean at baseline	Mean at 6 months	P value
NYHA class (mean ± SD)	3.0 ± 0.0	0.72 ± 0.85	< 0.005
CCS class (mean ± SD)	0.55 ± 0.82	0.1 ± 0.3	< 0.005
Resting LVOTG in mmHg (mean ± SD)	47 ± 36	12 ± 16	< 0.005
Provoked LVOTG in mmHg (mean ± SD)	88 ± 42	18 ± 25	< 0.005
Septal thickness in mm (mean ± SD)	30 ± 4.4	27 ± 4	< 0.005

NYHA – New York Heart Association; CCS – Canadian Cardiovascular Society; LVOTG – LVOTG = left ventricular outflow tract gradient; mm – millimeters

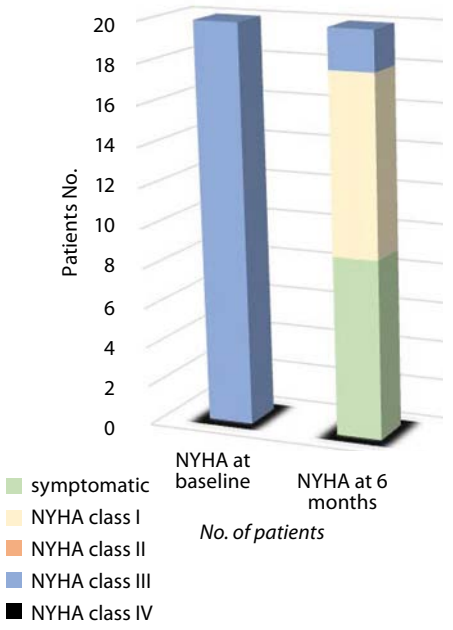
and at last clinical follow-up with NYHA class improved to (0–1) and, in those with baseline anginal symptoms, to CCS class 0–1. Symptomatic improvement was sustained in those patients without medications which were typically stopped 3–6 months after the procedure.

Six patients did not have immediate procedural success, three of them (both had immediate LVOTG reduction of less than 50 %) failed to get a reduction in LVOTG postprocedural and at 6 months follow up. One patient underwent a repeat ASA procedure 6 months later that had immediate and long-term successful results and the other two patients are planned for repeating ASA in the next few months. For the other 3 patients (all had immediate LVOTG reduction of more than 50 % but to more than 20 mmHg), they had their symptoms improved and their LVOTG reduced successfully at 6 months and on long-term follow-up.

Therefore, at 6 months and at long-term follow-up, 18 patients (90 %) had successful ASA procedures, pending repeating ASA procedures for the resting two patients. Beyond 6 months, the reduction in LVOTG and septal thickness was minimal and occurred only in 3 patients. No further reduction in septal thickness or LVOTG was noted after 1 year in any patient.

After discharge from the hospital, none of the patients who underwent ASA developed AAE, CVA, infective endocarditis, or new high-grade heart block on the follow-up that vary according to the date of the index procedure (table 5). There was only one death by non-cardiovascular cause.

Fig. 3. *Symptomatic improvement 6 months after ASA*



NYHA – New York Heart Association

The outcome of ASA procedures was comparable between young adults and middle-age adults as shown in table (6) and there was no association between the presence of extreme septal hypertrophy and effectiveness of ASA as shown in table (7).

Discussion

HCM is a common genetic cardiac disease (11). Most of the available data about outcomes of ASA procedures were from either single centers or registries in the form of retrospective, non-randomized studies (58–64). However, these studies represent a significant part of the evidence supporting ASA efficacy in well-selected patients with symptomatic HOCM. Therefore, observational analyses are very important. In this study, we are reporting our experience at a tertiary referral heart center in Iraq performing ASA procedures.

Our study involved 20 patients who had undergone ASA at Nasiriya Heart Centre over six years. The rate of ASA procedure in our center (3.33/year) was comparable to that in previous similar studies (59–61).

Thirteen patients (65 %) were young adults who had a comparable outcome of ASA to that of older adults as shown in table (6). Alcohol septal ablation was reported to be controversial in this age group because of the absence of long-term data on the late effects

Tab. 5. *Duration of follow-up*

Duration	No (%)
Death	1 (5)
Lost to follow up	2 (10)
6 months to 1 year	3 (15)
1 to 3 years	6 (30)
3 to 6 years	8 (40)
Total	20 (100)

Tab. 6. *Outcome of ASA in young adults as compared to older adults*

Age	No. of patients (%)	Successful ASA No. (%)	PPM implantation No. (%)
≥ 40 years	7 (35)	7 (100)	1 (14.3)
20- < 40 years	13 (65)	11 (84.6)	2 (15.4)
P value	0.27	0.95	

ASA – alcohol septal ablation; PPM – permanent pacemakers

Tab. 7. *Effectiveness of ASA in patients with and without extreme septal hypertrophy*

Septal wall thickness	No. of patients (%)	Successful ASA at 6 months No. (%)
≥ 30 mm	9 (45)	8 (88, 9)
< 30 mm	11 (55)	10 (90, 9)
P value	0.88	

ASA – alcohol septal ablation

of a myocardial scar (12). However, in a recent large study, Liebregts et al found that ASA in younger patients with HOCM was safe and effective for relief of symptoms at long-term follow-up and they propose that the indication for ASA can be broadened to younger patients (65).

Symptomatic improvement after ASA was found to be excellent in our study. Only two patients continued to have NYHA class III symptoms. Patients benefited from the early and sustained reduction in NYHA and CCS classes. In systematic reviews of 42 studies by Alam et al. and of 12 studies by Agarwal et al., a significant reduction in symptoms was reported after ASA with a significant reduction in both NYHA class and CCS class (50, 66).

The most pronounced effect of ASA occurs in the first 6 months, with the reduction in resting LVOTG, provoked LVOTG and septal thickness almost occurred during this time period. Ongoing minimal reduction was observed up to 1 year in only 3 patients. Two studies found that there is an ongoing reduction in LVOTG beyond 6 months (63, 59). Jason et al postulated that ongoing remodeling may be related to their procedure strategy by selecting septal perforator sub-branches that enable better targeting of basal septum [59]. However, two other studies postulated that LV remodeling reach a plateau (non-significant reduction in septal thickness and LVOTG) after 6 months from ASA (61, 64). In our study, patients' variation in their plateau time may be related to the variation of basal septum proportion that is supplied by the target septal perforators (80).

In our study, 2 patients failed to have procedural success immediately and at 6-month follow-up. Sorraja et al in their study found that patients with ≥ 3 characteristics (age ≥ 65 years, LVOTG < 100 mmHg, septal hypertrophy ≤ 18 mm, LAD diameter < 4.0 mm) had superior 4-year survival free of death and severe symptoms in comparison to those with two characteristics and ≤ 1 characteristic. The volume of alcohol injected, the number of arteries injected, or the size of septal perforator artery was not predictive of clinical success (82). Steggerda et al also found that the presence of a non-ablated proximal septal branch with a greater distance to the ablated septal branch, non-involved parameters in our study, is associated with an unsuccessful outcome after ASA (83). In our study, these predictors could not be applied statistically, because we have only 2 remaining patients with unsuccessful ASA procedures.

Nine patients in our study had extreme septal hypertrophy (septal wall thickness ≥ 30 mm). The success of ASA procedures was comparable in our study between patients with and without extreme septal hypertrophy as shown in table (7). This was in keeping with Yin-Jian Yang et al in their study who found that the effectiveness of ASA seems comparable between patients with and without extreme septal hypertrophy (85).

One of the major concerns of ASA is the arrhythmogenicity of the resultant LV myocardial scarring which is more in ASA compared to myectomy, as shown by Valeti et al in their study using CMR imaging for comparison (67). Three studies found, compared to myectomy, a small increase in AAE after ASA (68, 69). However, a recent meta-analysis by Liebregts et al had confirmed that there is no increased risk of AAE and long-term mortality after ASA as compared to myectomy [70]. In our study, no patient suffered any AAE within 1 year of the procedure.

Acute conduction disturbances after ASA likely occur due to ischemia of the conduction system. Transient inflammatory and edematous response to direct toxic effects of alcohol may be also responsible for transient postprocedural conduction disturbance. The most common conduction abnormalities after ASA was RBBB that occurred in 50 % of patients that was comparable to its incidence in our study which was 45 % (84). The reported incidence of PPM insertion after ASA varies from 2 to 35 % in large studies (71–73). Procedural techniques (e. g. selection of septal perforator sub-branches) might explain this variation (59). In addition, many factors had been shown to predict CHB after ASA like old age, high LVOTG, baseline and postprocedural ECG conduction disturbance (74–76). The volume of ethanol was not associated with postprocedural CHB in many large studies (59, 77, 78). In our study, 3 patients (15 %) developed CHB, all of them had intraprocedural CHB, none of them had baseline conduction disturbance on ECG. PPM implantation was significantly associated with intraprocedural CHB and marginally associated with higher baseline provoked LVOTG, while baseline resting LVOTG, alcohol amount and postprocedural RBBB were comparable between patients required PPM implantation and the others, as shown in table (3).

Our patients had their temporary pacemaker removed 48 hours after ASA and for more duration to patients with intraprocedural or postprocedural conduction disturbance. If still in CHB, a PPM was implanted generally within 5–7 days. Three major studies reported very low rates of PPM insertion by leaving temporary pacing wires in for up to 7 days, together with a strategy of non-implanting a PPM earlier than 14 days (60, 61, 79). However, all three patients in our study who had implanted PPM were found to have variable percentages of pacing (none of them had a zero pacing) at regular interrogation of their devices. Importantly, Fifer et al in their study found no association between PPM implantation and long-term mortality after ASA (72).

The only death in our study was by a non-cardiovascular cause. Multiple meta-analyses had shown that procedural mortality was comparable to that of surgical myectomy (49–52). In addition, Sorojja et al found that long term survival after ASA was comparable to that of the general population (45), while Kuhn et al and Veselka et al reported a significant decrease in in-hospital and long-term mortality when reducing the mean amount of alcohol injected per procedure without decreasing the procedures' efficacy (43, 81).

Limitations

The findings of this study should be interpreted in the context of its limitations which are:

- It is a non-randomized, descriptive single-center study with a relatively small number of patients.
- For all patients, medical treatment before ASA did not involve disopyramide.

Conclusion

Our data showed that, in carefully selected patients, alcohol septal ablation is a safe procedure with a low incidence of complications. Although our sample size is small to conclude procedural outcomes, we have demonstrated that ASA is technically feasible in most of the patients assessed, and is associated with high procedural success rates with low risk of non-fatal complications.

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