CARDIOMYOPATHY-TACHYCARDIA INDUCED? REVIEW

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Summary

Tachycardia-induced cardiomyopathy. if diagnosed and treated, in time is a reversible cause of heart failure. The clinical manifestations, neurohumoral mechanisms, and treatment of this condition are like any other type of heart failure. Uniquely, treatment of the tachycardia responsible for the condition, often results in clinical improvement and gross resolution of the heart failure. It is not known weather it scars the myocardium. which could increase the vulnerability to future development of heart failure but it is well known that once myocardium has dilated and thinned out, complete reversal to normality is not possible. The importance of restoring and maintaining sinus rhythm, or at least controlling the ventricular rate in these patients cannot be overemphasized. In patients with pacemakers, using demand mode pacing and sequential dualchamber pacing is important. Various standard therapeutic options can help contain the damage and probably reverse it. Treatment modalities of recent past like radiofrequency catheter ablation and pacing, ICDs and ventricular resynchronization have to be brought to the benefit of common people in terms of cost and availability. Further research in the field of causes and control of arrhythmias, apoptosis, fibrosis, remodeling, cardiomyocytes genesis and implants, may discover more pivotal and focused therapeutic targets for intervention. Until then, heightened awareness of this condition, possible prevention, timely and judicious use of available resources are only hope for its management.

INTRODUCTION

Cardiomyopathy is defined as the disease of the heart muscle of unknown cause. Congenital heart diseases, valvular heart diseases, hypertensive heart diseases and ischemic heart diseases can all cause dilated cardiomyopathy. These four groups of diseases are to be ruled out to start with. A large group of diseases recognized to cause secondary cardiomyopathy are then excluded to call it Primary cardiomyopathy. Professor J. Goodwin classified cardiomyopathies into three groups[1] dilated,[2] hypertrophic, and [3] restrictive. 1 All these types end up in dilated cardiomyopathy in the later stages. A WHO task force on cardiomyopathies proposed two groups [1] heart muscle disease of known causes (e.g. congenital, valvular, hypertensive, ischemia, metabolic, toxic, heredofamilial sensitivity and infectious diseases.;[2] Heart muscle disease of unknown causes to be termed as cardiomyopathies. [2] These diseases were further classed as dilated, hypertrophic or restrictive and defined as follows.

Dilated cardiomyopathy. "The condition is recognized by dilatation of the left or right ventricle or both ventricles. Dilatation often becomes severe and is invariably accompanied by hypertrophy. Systolic ventricular function is impaired Congestive heart failure may or may not supervene. Presentation with disturbances of ventricular, atrial rhythm is common and death may occur at any stage".

Hypertrophic cardiomyopathy. "This conditions is characterized by disproportionate hypertrophy of the left ventricle and occasionally also of the right ventricle which typically involves the septum more than free wall but occasionally is concentric. Typically, the left ventricular volume is normal or reduced. Systolic gradients are common. Inheritance is usually by an autosomal dominant gene with incomplete penetrance. Characteristic morphological changes, usually most severe in the septum, have been described."

"The clinical diagnosis of HCM is established most easily and reliably with two-dimensional echocardiography by demonstrating left ventricular hypertrophy (LVH) (typically asymmetric in distribution, and showing virtually any diffuse or segmental pattern of left ventricular [LV] wall

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thickening). Left ventricular wall thickening is associated with a nondilated and hyper dynamic chamber (often with systolic cavity obliteration) in the absence of another cardiac or systemic disease (e.g., hypertension or aortic stenosis) capable of producing the magnitude of hypertrophy evident, and independent of whether or not LV outflow obstruction is present". [3]

Restrictive cardiophyopathy." This may exist either with or without obliteration. Restrictive cardiomyopathy includes endomyocardial fibrosis and Loffler's cardiomyopathy (endocarditis parietalis fibroplastica). Endomyocardial scarring usually affects either one of both ventricles, and restricts filling, Involvement of the atrioventricular valve is common but the outflow tracts are spared. Cavity obliteration is characteristic of advanced cases".

It is recognized that occasional patients with heart muscle disease of unknown cause have morphological or functional abnormalities that do not allow classification in one of these three major groups. There also may be transition, over a period of time, from one type to another. Despite these problems, this simplified classification has merit and warrants wide adoption. It is hoped that future investigation may elucidate the cause or causes of these currently unknown disease states

Myocarditis is a close associate of this disease with extremely blurred margins between the two. By far the commonest form of this disease is then labeled as idiopathic cardiomyopathy after exhaustive work up with all the available modem tests. In the past literature thyrotoxicosis found a passing reference as a cause of this disease. Cardiomyopathy has been induced by performance enhancing drugs that included thyroxin in a body builder with normal coronaries as reported by Mark PB, et al. [4] The mode in these cases could have been tachycardia as it is now proven that tachycardia induced cardiac muscle disease is much more common then was realized in the past. There have been extensive studies on this subject regarding the mechanism and causes. There are two types of cardiomyopathies induced by tachycardia -- supraventricular and ventricular type.

ATRIAL TACHYCARDIA INDUCED CARDIOMYOPATHY SUPRAVENTRICULAR TYPE

Tachycardia induced cardiomyopathy was reported in-patient of atrial fibrillation in 1913 by Gossage et al.[5] Brill et al. reported another case of atrial fibrillation that presented with the heart failure and improved when the sinus rhythm was restored.[6] Experimental work was conducted on tachycardia induced cardiomyopathy by Whipple et al.[7] This disease can result by a variety of arrhythmias of supraventricular or ventricular origin. The faster the heart rate the earlier the induction of the cardiomyopathy. The earlier it has been seen within 24 hours after cardiac pacing at high heart rate.[8] The causative tachycardia my or may not be present at the time of heart failure. The ventricular dysfunction and its reversal are dependent on the rate and duration of the causative tachycardia.

MECHANISM

Atrial and ventricular tachycardia both can result in tachycardia-induced atrial cardiomyopathy. Atrial fibrillation can result in, atrial or ventricular type of cardiomyopathy and. success of restoration to sinus rhythm depends upon the duration of atrial fibrillation. [9] The patient can present with arrhythmia or heart failure associated with dilated cardiomyopathy.

Atrial Tachyarrythmia induced cardiomyopthy is defined as atrial pathology caused by the tachycardia. Atrial fibrillation is the commonest VT to cause this cardiomyopathy. In some cases AF, however, could be the result of dilated cardiomyopathy The basic change is dilatation of atrium and contractile dysfunction The molecular mechanism underlying these changes has been worked out recently. There is a fault in the L-type Ca channels. This results in increased Ca++ extrusion by the Na+/Ca2± exchanger.[10] The atrial cardiomyocytes cannot regulate calcium properly. This results in electrical disturbances due to excess calcium inside the cell. There is up regulation of Na+/Ca2± exchanger and down regulation of or altered function of the L- type Ca2+ channels .[11] The contractility of the remodeled atria is depressed as a result of increased Ca2+ extrusion by the Na+/Ca2± exchanger. In this way

atrial cardiomyopathy differs from the ventricular cadiomyopathy induced by the tachycardia, where the fault is down regulation of beta-receptors. changes in ECM and structural proteins due to chronic stimulation. Atrial fibrillation was induced by prolonged rapid ventricular pacing in an experimental model by Li. et al. where a reduced atrial transient outward Ca2+(lto) and L-type Ca2+ current, slow delayed rectifier K+ current and increased Na+/Ca2±exchanger activity was observed. [12]

The Ace inhibition reduces angiotensin II leading to decrease in MAPK expression and reduction in phosphorylated form of c-Jun N -terminal kinase.[13] It has been shown that ACE inhibition attenuates heart failure induced fibrosis of atrium and remodeling microscopically and echoardiographically respectively, and halts AF progression.[14] The fibrosis may not reverse completely as angiotensin 11 hence, atrial fibrosis is mediated by some other pathways as well.[15,16] Atrial tachycardia induced cardiomyopathy differs from its ventricular counterpart due to cellular changes This has been proved in studies assessing apoptosis, fibrosis, white cell infiltration and cell death and angiotensin II concentration. There has been more rapid rise in angiotensin 11 concentration, more tissue apoptosis, inflammatory cell infiltration and cell death in the atrium as compared to ventricle. High levels of transforming growth factor -B may be mediating fibrisis.[17] Treatment of atrial tachycardia induced cardiomyopathy should include ACE inhibitors. Cha et al observed complete recovery of ionic remodeling but persisting structural changes in the atrial tissue while studying the mechanism of reversal of these changes.[18]

VENTRICULAR TACHYCARDIA INDUCED-CARDIOMYOPATHY

The chronic ventricular tachycardia is the main cause of Techy-Induced cardiomyopathy with the chronic SW being the cause in other cases. The single chamber ventricular pacing comparable to VT is the worse than the sequential duel chamber pacing, atrial paced, that is comparable to SVT.[19] These changes occur quickly due to chronic and persistent abnormal ventricular activation as compared to intermittent ventricular tachycardia. [21]

ANATOMICAL AND FUNCTIONAL CHANGES IN THE VENTRICLE. (GEOMETRY)

Chronic stimulation of the ventricular myocardium increases the LV area resulting in mitral annular dilatation, alerted ventricular geometry causing mitral regurgitation which reduces effective cardiac output, leaving elevated LV end-diastolic pressure thus increasing stretching upon the myocardium muscle and ultimately dilating the LV.[20,22]. The mitral regurgitation causes further LV dilatation and accelerates the vicious circle. Other mechanical changes like loss of LV torsion and increased LV stiffness also contribute towards the impairment in LV function and increased dilation of LV.LV torsion during systole reduces the transmural strain on the muscle fiber and the recoil in diastole enhances LV filing. Abnormal LV torsion causes inefficient contraction and deranged elastic properties by stiffening the ventricle, both result in inefficient expenditure of energy and worsens the LV diastolic function.[23] The cardiac structural proteins, extra cellular matrix and proteoglycans are the underlying abnormalities.[24] Tachycardia induced cardiomyopathy reverses after treatment of the causative tachycardia. This reversal may be partial and late, as the improvement is guided by the gross LV contractility. The microscopic changes may or may not reverse.[25]. Spinale et al has shown in dogs that recovery from tachycardia induced cardiomyopathy improves pump function mediated through the reduction in LV hypertrophy rather than the normalization of LV geometry and myocytes contractile funtion.[26] This could also be due to genetic influences as evidenced by increase in mRNA expression. This may be due to cardiac memory that turns on the compensatory switch for LV hypertrophy and does not switch of, with the restoration of the sinus rhythm.

DISTURBANCE IN THE STRUCTURE AND FUNCTION OF THE CARDIOMYOCYTE

Chronic ventricular stimulation increases ventricular mass by recruiting adult myocardiocytes to divide and proliferate. Jovanovic et al using laser confocal microscopic cross section images of the myocardium has shown increased number of cells in both longitudinal and transversal sections.[27] The ACE

inhibitor enalapril abolished these changes. This proves that hyperplasia due to chronic stimulation is a possible mechanism of cardiac chamber enlargement. It also shows that potential to reversibility should be evailed early by the treatment with the ACE inhibitors. The other mechanism proposed are changes in capillary structure, function, distribution and increased capillary -myocyte distance (in the LV myocardium.[28] These changes may impair MBF and limit Oxygen delivery to the myocardium thus worsening the LV function. Myocardial blood flow is reduced and coronary vascular resistance is also increased in this disease.[29] The reduced coronary blood flow and reserve has also been observed in the hibernating myocardium, caused by the ischemia which reverses after the improvement of blood flow to the ischemic myocardium. A hibernating myocardium could also may exist in this disease as it improves after the restoration of the normal sinus rhythm. Myoglobin deficiency has also been observed in experimental canine model of pacing induced cardiomyopathy.[30]

ABNORMALITIES IN THE SINGLING AND NEUROHUMORAL PATHWAYS.

There is over activity of rennin angiotensin aldosterone axis causing abnormalities in sodium handling and increased levels of atrial natriuyretic peptide (ANP) in tachycardia induced cardiomyopathy while the vasodilator, natriuretic and rennin-lowering effects of ANP are damped in the canine model of tachycardia induced cardiomyopathy. This could be due to blunted cyclic GMP expression in response to the ANP.[31] Abnormal sodium handling has very important role in these neurohumoral changes. The ANP level in the atrial tissue is reduced while the sodium level of ANP is high in the experimental models. Brain levels of ANP are also increased although to a lesser degree than ANP.[32] This ANP release is stimulated by the increased heart rate, high right atrial pressure and increased atrial volume. ANP release is reduced as the heart failure advances perhaps due to depletion in the ANP stores.[33.34] Increased levels of angiotensin II and aldosterone cause myocardial fibrosis in the heart failure. This may be the reason why ACE inhibitors and angiotensin receptor blockers are beneficial in this disease.

Reduced baroreflex sensitivity has been seen in this disease, which reverses to normal quickly with the restoration of the normal sinus rhythm.[35] There is increased nor epinephrine, reduced cardiac nor-epinephrine uptake-1 carrier sites density.[36] Elevated endothelien-1 (ET-1) level has been reported to accelerate the progression of the tachycardia induced cardiomyopathy. ET-I produced mitochondrial changes are reversed using ET-IA a receptor blockage.[37] These are the potential factors for intervention in the future treatment of this disease.

ELECTRICAL ABNORMALITIES

Dilated cardiomyopthy provides a perfect background for electrical abnormalities. There is severe electrical heterogeneity in the myocardium. The abnormal depolarization of the dilated, fibrosed myocardium. due to prolonged action potential is responsible for the ventricular arrhythmias. The association of prolonged depolarization with increased dispersion and its contribution towards bradycardial dependent ventricular arrhythmia has been studied in a porcine model of tachycardia-induced cardiomyopathy.[38] They observed that Tic intervals were prolonged, transmural gradient was reduced and spatial dispersion of repolarization was reduced in this model. There was reduced current density in the potassium channels in the subendocardium. They however concluded that repolarization was uniformly prolonged in this model and no predisposition to bradycardial dependent arrhythmias was found. Another study was conducted by Pak. et al into sudden cardiac death SCD in congestive heart failure in porcine model of tachycardia-induced cardiomyopathy.[39] They suggested that the cardiomyopathy. exhibited malignant arrhythmias and SCD through repolarization abnormalities; they suggested that cellular hypertrophy modulates mechanosensitive i.e. stretch -activated channels. Stretch activated channels are a distinct group of cardiac ion channels located on the outer membrane of the cell that permits cation movements into and out of the cell. They contribute towards the reversal potential (the membrane voltage at which the direction of the current flow through an ion channel changes direction) during diastole. The other group of ion channels are cell volume activated channel. (Activated by increase in the cytosolic volume) It has been observed that the development of

the cardiomyopathy. alters the set point for and causes persistent activation of swelling - currents.[40] These diastolic stretch receptors, in general, result in cardiac depolarization. However in the CHE, this diastolic stretch is non-uniform and results in electrical hetrogenicity, setting a stage for arrhythmias. Propensity for ventricular arrhythmias in patients with heart failure has been proposed to be due to remodeling of important K+ and Ca2+ currents in cardiac Purkinje cells, resulting in reduced repolarization delay. The slowing of the L-type calcium current inactivation was observed during this experiment. [41]

ABNORMALITIES IN THE MYOCARDIAL EXTRA CELLULAR MATRIX.

Extra cellular Matrix ECM supports the cellular network of myocardium. When there are abnormalities in the ECM LV stiffness increases and contractility is impaired. Spinale et al examined these changes in ECM in tachycardia-induced cardiomyopathy, during its development and regression.[42] There was a decrease in collagen concentration, increase in salt extractable collagen, myocyte adhesion to basement membrane component was reduced and chondrotin sulfate concentration was increased during tachycardia. All the changes improved after tachycardia stopped.

The LV dysfunction, both systolic and diastolic, is seen in dilated cardiomyopathy. Titin, a structural myocardial protein, is responsible for myocardial stiffness and diastolic ventricular relaxation. There are two titin is forms with different stiffness characteristics in the myocardium. the stiff N2B isoform and more compliant N2BA isoform. The changes in titin isoform expression have been proposed as a mechanism of diastolic dysfunction seen in this form of cardiomyopathy. [43]

Hypertension is commonly associated with diastolic dysfunction which is an active process. In a rat model of hypertension altered titin expression has been reported.[44] The same abnormal titin switch has also been observed in the ischemic left ventricular diastolic dysfunction in the human heart as well.[45] Diastole expends ATP for its execution and less supply of ATP could lead to diastolic dysfunction. Impaired ATPase activity of the sarcoplasmic reticulum leading to impaired Ca 2+ uptake and release has been reported in systolic and diastolic dysfunction.

ABNORMALITIES IN EXCITATION-CONTRACTION COUPLING

There are electrophysiological abnormalities in the cardiomyocytes and structural changes in the myocardium. of Tachycardia-induced cardiomyopathy. L-type Ca2+ channel dysfunction plays an important role in the observed electrical abnormalities.[46] the studies in a canine model has shown T-tubule system and L-type Ca 2+ channels abnormalities.[47] The failing cardiomyocytes demonstrated a significantly lesser regularity of the T-tubule system and a relative loss of T-tubules. There is unchanged calcium ion channel density with decreased charge attributable to L-type Ca2+ channels in the failing myocytes. The cardiomyocyte dyscontractility due to change in geometric orientation and basement membrane disruption causes LV dysfunction.[48] There is increase in sympathetic tone, down regulation of the cardiac b-adrenergic receptors, reduced b -receptor transduction, and decreased adenylate cyclase activity.[49] The paced hearts have increased number of ventricular and atrial myocyte nuclear apoptosis, reduced levels of Bcl-2 (an inhibitor of apoptosis), elevated Fas and Fas-L, and elevated caspase-2 and caspase-3,

ROLE OF OXIDATIVE STRESS AND INFLAMMATION

Oxidative stress, resulting from high rates, causes mitochondria oxidative damage The mitochondria DNA is more vulnerable to oxidative damage than nuclear DNA. An imbalance between pro-oxidant and anti-oxidant pathways is probably an explanation for this observation. Xanthine oxidase is pro-oxidant enzyme and its inhibition in failing hearts improves cardiac efficiency probably due to reduced oxidative stress. Nitric oxide synthase (NOS), being an anti-oxidant enzyme, is cardio protective. Saavedra et al.[50] showed, in a canine model, that both these signaling systems participate in regulating efficient myocardial contractility and up regulation of the xanthine oxidase is responsible for the mechanical-energetic uncoupling in heart failure. The myocardial

contractiliy improves with antioxidant therapy, possibly as a result of tipping the balance in favor of nitric oxide synthesis.[51].

Selegiline, an inhibitor of monoamine oxidase, has also been shown to have antioxidant and anti-apoptotic effects. It has been shown by Qin et al.[52] in the rabbit model of heart failure that rapid cardiac pacing increased plasma nor epinephrine, cardiac oxidative stress, and myocyte apoptosis; reduced Bcl-2 and the Bcl-2 to Bax ratio (in favoring apoptosis); and decreased LV fractional shortening and dP/dt and reversed all the observed changes with selegiline. They suggested that this action on myocyte apoptosis was related to reduction in oxidative stress and restoration of the balance between apoptotic and anti-apoptotic proteins. Although literature on human subjects is limited, it seems to support that oxidative stress does play a role in vivo as well. Malondialdehyde (MDA), a marker of lipid peroxidation and has been used as a marker of oxidative stress. Velez et al.[53] measured plasma levels of MDA in patients with varying severity of CHF and in control subjects. They showed that in symptomatic patients with chronic CHF, there was significant correlation between MDA levels and chronicity of CHF These studies support the use of anti-oxidants heart failure.

Tachycardia-induced cardiomyopathy. was produced in dogs, with cardiac pacing to study role of Inflammation. There was an increase in myocardial monocyte infiltration, monocyte chemo attractant protein-1 expression, and renin-angiotensin system and local matrix metalloproteinase activity. The loss of cardiomyocytes (apoptosis) was observed during progression of heart failure as a result of various ongoing metabolic and neurohumoral insults. The increase in apoptosis correlated with elevated epinephrine levels.[54] In cardiomyopathy. of recent onset, increased expression of Fas and TNFR1 was associated with minimal recovery of LV function. Apoptosis limits myocardial recovery, and represents a potential target for therapeutic intervention.[55]

CLINICAL DIAGNOSIS AND MANAGEMENT

Diagnosis

Tachycardia induced cardiomyopathy. usually presents with signs and symptoms of heart failure. A very high index of suspicion is required to diagnose this disease. It can start at any age. It has been reported in fetal age where it results in hydrops fetalis. [56]

Tachycardia induced cardiomyopathy is invariably associated with tachycardia which may be taken as a result rather than a cause of this disease. It is important to establish whether the tachycardia or cardiomyopathy. started first. A detailed and precise history is essential requirement to save the diagnostic plan going astray. The condition should be suspected in a patient with clinical or investigational evidence of heart failure in the setting of heart muscle disease preceded by the supraventricular or ventricular tachycardia. Electrocardiogram and echocardiogram, although cheap, are the most valuable investigations. It has to be realized that tachycardia some times can be paroxysysmal, asymptomatic or long gone, as a cause, to return as a result of heart failure. Holters monitor, cardio memo, telemetry and real tracker can be of help in such circumstances. There is no particular rate above which this condition occurs. Other causes of impaired left ventricular function, particularly ischemic heart disease has to be excluded to make this diagnosis. It has been observed after 24 hours of fast ventricular pacing in animal experiments, progresses for 3-5 weeks, starts improving within 48 hours of cessation of the pacing and LV function gets back to normal or near normal by 1-2 weeks. There is ample evidence in the literature that it does not reverse completely in all cases. This condition can result from all kinds of supraventricular and ventricular tachycardias.. Cardiomyopathy is more severe with VT than SVT. AF is very frequently associated with tachycardia induced cardiomyopathy. AF can be involved in a vicious circle. AF can cause or result from ventricular failure. Left ventricular function impairment can be diastolic due to reduced LV filling due to loss of atrial kick or systolic, caused by impaired contractility due to changes in the myocardium. as a result of chronic stimulation of the ventricle at high rate.[57] AF, hypertension, ischemic heart disease and heart failure are frequent companions of old age. It may be difficult but essential, to point out which started the other. Conversion of AF to sinus rhythm or ventricular rate control improves ventricular cardiomyopathy.

Management

The management of tachycardia induced cardiomyopathy. includes eradication of the causative arrhythmias and control of heart failure till such time that ventricular myocardium. takes care of itself. A large number of agents are available for rate control in AF. The combination of digoxin and atenolol has been reported as most effective for rate control for AF.[58] However, there is a case for restoration of sinus rhythm in patients with AF. It has been shown that cardio version provides further benefit beyond rate control.[59] Rhythm control is not superior to rate control as shown by the AFFIRM trial.[60]

1-Arrhythmias

Underlying cause of tachycardia has to be addressed. The systemic causes of tachycardia like dysthroidism should be investigated and treated. The cardiac arrhythmias should not only be documented but properly classed as well because different treatment is required for different arrhthmias. Treatment can be

A-Medical

Drugs are used for rhythm control and rate control. In patients without ischemic heart disease, Class IA and Class IC agents are often useful in treatment. B-Blockers like Sotolol, Bisoprolol and calcium channel blockers like verapamil (Classes II and IV, respectively) are effective and most commonly used for ventricular rate control in AF and prophylaxis of SVT. They are also the drugs of choice for idiopathic VT such as RVTO tachycardia, the vast majority of which are eAMP-dependent. Sotalol and amiodrone (Class III) have also been used to treat SVT and VT. Chen et al.[61] reported the use of amiodrone in fetal life to treat PJRT. Flecainide has also been reported as effective in fetal life.[62] Digoxin is used mostly for rate control, but some patient of AF and Atrial flutter do convert to sinus rhythm due to improvement in homodynamic mileu. Adenosine and verapamil is useful for acute SVT and lignocaine for acute VT. In cases resistant to these drugs, amiodrone, can be used both for SVT and VT. Propafenone is very effective for VT.

B-Electrical Cardio version

This mode of treatment is mainly used in acute and emergency cases, in life threatening arrhythmias like VF, VT and VT. Cardio version is also used for restoration of sinus rhythm in atrial fibrillation and flutter. The atrial defibrillator has been reported to revert tachycardia-induced card cardiomyopathy.[63]

C-Ablation and Pacing

This is mainly used for SVT. The electrical currents have given way to much safer radiogrequency ablation, with an impressive success rate exceeding 95%. Pulmonary vein isolation and accessory pathway ablation are very commonly employed now a days. This can result in AV block and a ventricular pacemaker is usually required. A return of arrhythmias is common with these procedures. The "ablate and pace" modality of treatment is superior to pharmacological management.[64] The Australian Intervention Randomized Control of Rate in Atrial Fibrillation Trial (AIRCRAFT)[65] reported that at 12 months follow-up, there was no significant difference in LVEF or exercise duration on treadmill testing; however, the peak ventricular rate was lower in the AV node ablation and pacing group during exercise and activities of daily life. Patients in the AV node ablation and pacing group had fewer symptoms at 6 and 12 months. The ablation and pacing group reported a 6% better quality of life at 6 months.

D-Intracardiac Defibrillators(ICD)

Intracardiac defibrillators are programmed devices that detect arrhythmias and deliver synchronized or nonsynchronised shock to restore the normal sinus rhythm. These devices have proved life saving in malignant and incessant arrhythmias like, ventricular tachycardia, ventricular fibrillation and atrial fibrillation. The cost is prohibitory for use in this part of the world. Average cost per device with annual follow up of single patient is about 62 thousand US dollars.

E-Surgery

Surgical ablation is usually carried out for SVT during cardiac surgery for other indications. Maze and corridor operation are now rarely required with much safer radiofrequency ablation available.

2-TREATMENT OF HEART FAILURE

A-Medical

Heart failure can be effectively managed using standard, very effective medications. Control of fluid volume with the use of diuretics, ace inhibitors, AR Blockers, reduced fluid intake and, 02 inhalation are required in acute situation. Nitrates can be used for

the redistribution of the fluid volume in severe cases till diuresis has taken effect. Digoxin and ventricular assist devices can be used in exceptionally resistant case. Low dose beta-blocker help by dampening the exaggerated sympathetic response to the failing heart. Probucol has been used to combat oxidative stress in the dog model of tachycardia-induced cardiomyopathy.[66] It also had favorable neurohumoral effects. Carvedilol is one of the newer b-blockers with prominent anti-oxidant and anti-activity apoptotic. This has been demonstrated in the experimental studies.[67,68] In addition, carvedilol has also been shown to have antiinflammatory activity. [69] A recent pilot study by Chin et al.[70] showed that b-blockers, but not ACE inhibitors, reduced lipid per oxidation in patients with CHF. Carvedilol may therefore be superior to other b-blockers in the management of patients with heart failure. Any precipitating causes like anemias, infections and renal impairment are to be sought and treated.

B-Resynchronization

Dilated cardiomyopathy. exhibits dysynchronus ventricular contraction resulting in reduced ejection fraction. Biventricular cardiac pacing has been used to resynchronize the cardiac contraction and proved useful in patients with ejection fraction of <30%. The cost again is enormous like ICDs. A new device with duel function of ICD and biventricular synchronized pacing is now available in the market with additional cost.

C-Rehablitation

Gradual exercise increase is another way of improving effort tolerance. It is best provided in cardiac rehabilitation programmes. Cardiac rehabilitation not only improves the functional capacity but also has been shown to improve survival. Koelling et at have reported improved clinical outcome and better self care adherence after one hour nurse educator delivered teaching.[71]

D-Myoblast Implants

Skeletal Myoblasts has been implanted into myocardium. Myoblasts taken from their own thigh muscles were cultured and expanded for 4-6 weeks and then either implanted epicardially improving EF from 22.9% to 34.6% [72] or delivered to the endocardial surface of the ventricle using a percutaneous, fluoroscopy guided, implantation technique. These patients showed improvement in EF from 34.4% to 36.6% and wall motion score improved 3 to 27. Na et at have shown peripheral granulocytes infused intracoronary to have improved EF from 48.9% to 56.7%. [73]

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