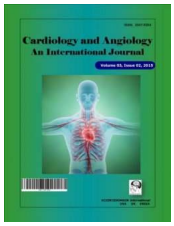

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Type 2 Diabetes and Myocardial Infarction in Central India: A Study and Review

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Author's contribution

The sole author designed, analyzed and interpreted and prepared the manuscript.

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ABSTRACT

Background: Clinical experience in specific geo-demographic contexts in diabetes and myocardial infarction (MI) deserves examination in the context of current medical knowledge and redefinition for enlightened evidence based medical practice.
Aim: Study aims to examine regional significance of known risk factors in incidence and outcome of MI in diabetics in comparison with non-diabetics.
Methods: Cases of myocardial infarction managed over 18 month period at medical college setting in central India in 35 to 75 year age range and free from major systemic co-morbidities were comparatively studied by categorizing as diabetic and non-diabetics. Demographic, clinical and laboratory information as well as complications and outcome profiles were assessed.
Results: MI in diabetics occurred at younger age, was common among women, overweight subjects and those with positive family history of ischaemic heart disease. Smokers and hypertensive's had high prevalence in MI cases among non-diabetics group. Poor glycaemic control and dyslipidaemia were common features in diabetic MI that was largely anterior suggesting extensive coronary atherosclerosis. Although statistically insignificant, most post infarct complications were more frequent among diabetics. Hospital stay was significantly longer for diabetic MI cases.
Conclusion: Study observations emphasize preventive role for dietary and lifestyle modification,

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weight reduction in diabetics and deterrence of smoking in non-diabetics as crucial. Management of hypertension is a necessary measure while good glycaemic control and correction of dyslipidaemia is pertinent in diabetics to reduce incidence and possibly severity of MI relevant in central Indian context. Angiotensin Converting Enzyme (ACE) inhibitors, beta adrenergic blockers and statins besides emergent glycaemic control with insulin have particular therapeutic relevance in diabetic MI.

Keywords: Type 2 diabetes; myocardial infarction; ischaemic heart disease; dyslipidaemia.

1. INTRODUCTION

Prevalence of coronary artery disease (CAD) has increased in parallel with urbanization and unhealthy lifestyle [1]. In India CAD prevalence has doubled over the past decade [2]. Half the deaths under 70 year age are attributed to CAD, which is projected to double in prevalence by 2030 [3,4]. Tobacco consumption, obesity, hypertension, diabetes and dyslipidaemia promote the core causal coronary artery pathology, atherosclerosis. Mental stress also aggravates cardio-metabolic risk [5]. Diabetes mellitus is a strong risk factor for CAD [6]. People with type 2 diabetes have increased risk of suffering MI that is more frequently of severe kind [7]. Mortality of acute myocardial infarction (MI) in diabetes is double that seen in non-diabetics [8].

1.1 Diabetes and Cardio-metabolic Risk

Possible mechanisms that increase risk of cardiovascular events in diabetes, include insulin resistance, changes in endothelial function, dyslipidaemia, chronic inflammation and release of mediators of inflammation, procoagulability and impaired fibrinolysis. Increased glycosylated haemoglobin (HbA1c) profile reflects cumulative long term bearing over macrovascular disease strongly determining cardiovascular outcome [9]. Postprandial hyperglycaemia accelerates atherosclerosis and has acute impacts on thrombus formation, endothelial function and intracardiac specialized conduction system [10-12]. Postprandial hyperglycaemia is thus linked to risk of cardiovascular events [13,14]. Both hyperglycaemia and sulfonylurea group of hypoglycaemic agents inhibits ischaemic preconditioning that occurs with ischaemic episodes as added reason of vulnerability of diabetics to myocardial infarction [15].

Abnormalities of lipoprotein metabolism are crucial to about 50% population attributable risk of cardiovascular events [16]. Insulin resistance elevate plasma insulin while stimulation of glucose uptake is deficient and promotes rise in triglyceride (TG) and decreased high density lipoprotein cholesterol (HDL-C) concentration, which constitute atherogenic risk [17]. Insulin resistance and hyperglycaemia manipulate lipid metabolism toward formation of atherogenic lipids. Insulin sensitive lipase is uninhibited causing increased formation of small dense low density lipoproteins, sdLDL [18,19]. There is increased release of non-esterified fatty acids from fat cells providing more triglyceride for production of very low density lipoproteins (VLDL). sdLDL has high transepithelial penetration and susceptibility to oxidation; reduced affinity for clearance through LDL receptor and high binding affinity to intimal proteoglycan. These contribute much more to formation of pro-aggregatory and vasoconstrictor mediators [20,21].The atherogenic lipids cause activation of inflammatory signaling pathways, such as nuclear factor kappa-B, MAP kinase and protein kinase-C; have direct effect on vasculature; cause oxidative/mitochondrial stress and genomic stress [22,23]. Increased TG and LDL-C increase sdLDL formation. TG plasma levels >135mg/dl (1.5 m mol/l) is indicative of significant increase in sdLDL formation and raised TG/HDL-C concentration ratio indicates the coronary disease risk [24].

Insulin resistance (IR) and consequent hyperinsulinaemia increases sympathetic nervous activity and salt sensitivity. Both these may promote development of hypertension. There is 2 fold higher prevalence of hypertension among the diabetics as compared to non-diabetics [25]. Mononuclear cells from insulin resistant individual more avidly adhere to the endothelium where hyperinsulinaemia causes increased expression of adhesion molecules. IR also increases endogenous inhibitor of endothelial nitric oxide synthase, asymmetric dimethyl arginine, and endothelial dysfunction is promoted [26]. The atherogenic lipid perturbations cause procoagulable state. Plasma plasminogen activator inhibitor-I (PAI-I) is increased in diabetes and decreases fibrinolytic activity. These changes relate to both vascular

pathology and occurrence of myocardial infarction.

Nephropathy indicated by microalbuminuria positively correlates with the extent of coronary artery disease in diabetics as well as in non-diabetics [27,28]. CKD defined as eGFR <45ml/min/1.73m^2 carries risk of myocardial infarction even greater than diabetes. CKD has been considered coronary heart disease equivalent [29]. Chronic kidney disease(CKD) worsens insulin resistance and dyslipidaemia [30]. CKD in diabetes further increases risk of myocardial infarction [31]. Increased renal dysfunction in diabetes and hypertension increases cardiovascular and overall mortality [32,33].

1.2 Myocardial Infarction in Diabetes: Understanding and Outcomes

Diabetes independently increases risk of death in MI by 57% [34]. More extensive coronary disease, other end organ dysfunctions and additional risk factors are all contributory [35,36]. Metabolic perturbations result in decreased ATP production, generation of oxygen free radicals, increased myocardial oxygen consumption and contractile dysfunction. Endothelial dysfunction impairs coronary perfusion at microvasculature level and ischaemia [37]. Heart failure consequent to MI results in tissue hypoperfusion and hypoxia, triggering xanthine oxidase activation. This results in generation of reactive oxygen species (ROS), inducing vicious circle [38,39]. The impaired compensatory mechanisms can increase infarct size and cause further impaired left ventricular function. Remodeling of the left ventricle consequent to myocardial infarction is a time dependant phenomenon. Within 3 hours of infarction, increases in end-diastolic and end-systolic volumes may be seen, which major predictor of clinical outcome is. In diabetes many specific changes relating heart affect overall myocardial remodeling. Subclinical diabetic cardiomyopathy with diastolic dysfunction may proceed before myocardial infarction setting systolic dysfunction. Hypertension supervening upon diabetes exaggerates cardiac fibrosis, than either condition alone [40]. Half the subjects of diabetic coronary artery disease also bear cardioautonomic neuropathy [41]. Autonomic neuropathy in diabetes increases vulnerability to circadian hemodynamic changes and ischaemia [42]. Myocardial infarction in diabetics associates higher heart rates indicating excess sympathetic and/or renin-angiotensin system activation, compared to that in non-diabetics.

Patients with type 2 diabetes and MI are heterogeneous lot, differing in degree of advance of coronary atherosclerosis, co-morbidities, clinical status at baseline, and metabolic profiles. Several studies indicate that effect of diabetes on cardiovascular (CVS) manifestations differ according to measured specific outcome. There is increasing evidence that CVS disease risk in diabetes may be reduced and must be identified and addressed, beyond key concern for glycaemic control. Combining of clinical course to profile of known risk factors increases insight to issues relevant to personalized prevention and management [43]. Studies are needed that generate clinical evidence by focusing on profile of pathophysiology, hierarchy of risk factors, clinical spectrum, management and outcomes in myocardial infarction in diabetes patients. The present study attempted an appraisal of clinical profile and course of MI among type 2 diabetics and non-diabetic patients in our specific region in central India to support evidence based clinical practices for prevention and management.

2. PATIENTS AND METHODS

The study included MI cases admitted consecutively in medical wards of Index medical college, Indore, between January 2015 to June 2016. Patients aged between 35 years to 75 years of either sex were included. Patients with antecedent or subsequently diagnosed with systemic diseases unrelated to MI were excluded. Of the finally studied 132 cases 69 were type 2 diabetics either known or diagnosed during hospitalization as per American Diabetes Association 2010 criteria e.g. Glycosylated haemoglobin HbA1c level >6.5 g/dl [44]. 63 cases of MI were found non-diabetic as per same criteria. Glycosylated hemoglobin (HbA1c) levels were also determined once upon admission. Diagnosis of MI was based on patient meeting at least 2 of following 3 criteria viz. typical chest pain/discomfort lasting at least 30 minutes; an electrocardiographic display of Q wave and/or ST segment elevation of>2 mm in at least 2 pre-cordial leads and abnormal rise of creatinine kinase MB levels indicating myocardial necrosis [45].

The study protocol was approved by college research ethics committee (IMCH/Res/Med-4/14). Patients consent was obtained after informing intent and nature of the observational

study for using information details of his/her condition and care for research, without ever disclosing personal identity. Various risk factors for coronary disease including age, gender, past MI, family history, physical activity profile, fresh fruit consumption, smoking habit, obesity (BMI >25), hypertension(BP >140/90 mm Hg), atherogenic dyslipidaemia triglycerides (TG)/high density lipoprotein cholesterol (HDL-C) ratio >3.5, glomerular filtration rate (60 ml/min/1.73 m^2), plasma malonaldehyde level and plasma total antioxidant capacity were examined and compared between the non-diabetic and type 2 diabetes victims of MI.

Family history of premature atherosclerosis was based on history of acute myocardial infarction (MI) in parent or siblings males under 55 and/or female less than 65 years of age. History of diabetes, hypertension or cerebrovascular disorder irrespective of age constituted positive family history. BMI was calculated as ratio of body weight to height squared and >25 kg/m^2 was taken as indicative of obesity in our patients [46]. Glomerular filtration rate was estimated from serum creatinine levels [47]. Physical activity status was assessed using relevant questionnaire. People with minimum 30 minutes continuous active exercise on at least 5 days in a week were deemed physically active and below that as inactive [48]. Fresh fruit intake was enquired as kind of fruits and amounts consumed over past month and past week. An average of at least one fresh fruit (about 200 g) consumed alternate day was taken as positive and less as negative [49].

Standard procedures were used for estimation of blood glucose, creatinine, and lipid profile as previously described [50]. Plasma malonaldehyde level was determined as thiobarbituric acid reactive substances [51], as indicator of oxidative stress. Total plasma antioxidant capacity was determined by FRAP (ferric oxide reducing ability of plasma) method [52]. Infarct localization was assessed from ECG pattern. Over the period of hospitalization, adverse cardiac events as ventricular and atrial arrhythmias, cardiogenic shock, complete A-V conduction block, cardiac failure were monitored and instances of death recorded.

Relative frequency distributions of subjects in two compared groups in respect to every parameter were analyzed. Chi square test and when necessary (for small cell numbers), Fishers exact test statistic were used.

3. OBSERVATIONS AND RESULTS

Prevalence of various assumed demographic and clinical risk factors in the two compared groups of MI cases is shown in Table 1.

The distribution of patients in two compared groups with respect to biochemical risk indices is presented in Table 2.

Significantly high proportion of diabetic MI cases were younger under median age of 62 years compared to non-diabetic group and significantly higher proportion of MI cases of female diabetic than non-diabetic .Significantly higher proportion of diabetic MI cases were obese (BMI>25), compared to the non-diabetic. There was no difference in history of past MI but positive family history of coronary disease was significantly more reported in diabetic MI group. Prevalence of physical inactivity or fresh fruit consumption was not significantly different among compared groups. Proportion of smokers and hypertensives were significantly higher among the non-diabetic MI group compared to the diabetic MI group patients.

HbA1c values indicating chronic hyperglycemic state implying linear relation to coronary risk [53], never reached 7.5g/dl in non diabetics but 44% of diabetic MI cases had higher than this level. Atherogenic dyslipidaemia had high prevalence in both groups with significantly greater prevalence among diabetic infarction cases. Nearly half the MI cases in either groups had eGFR lowered under 60 ml/min/1.73 m^2; and median value of plasma malonaldehyde was elevated to 3.84 µM/L, i.e. double the normal 2 µM/L. Median plasma FRAP was reduced to half relative to normal 1000 µM/L. These uniformly heightened parameters did not have significant differences in prevalence in the two groups.

Table 3, presents profile of complications and outcome among the two studied groups of MI patients. Cardiogenic shock is seen in less than 5% cases in either group. Ventricular fibrillation occurred exclusively in 6 of the 69 diabetic MI cases as significant difference from the non-diabetic group. Other cardiac complications e.g. Atrial fibrillation, atrioventricular (AV) conduction block and pulmonary edema were also more frequent among diabetics but the differences were not statistically significant. Diabetic MI cases frequently required longer hospital stay beyond 8 days. Death rate was not significantly different though marginally more in diabetic MI.

Table 1. Distribution of patients studied in the two groups with reference to defined demographic and clinical risk factors. Percentage of values are shown in parenthesis

	Non-diabetic MI (n=63)	Type-2 diabetic MI (n=69)	P
Age (Years)			
>62	35 (55.5%)	26 (37.7%)	<0.05
≤62	28 (44.4%)	43 (62.3%)	--
Gender			
Male	52 (82.5%)	46 (66.7%)	<0.05
Female	11 (17.5%)	23 (33.3%)	--
BMI			
>25	28 (44.4%)	43 (62.3%)	<0.05
25 and <	35 (55.5%)	26 (37.7%)	--
Past MI			
Yes	2 (3.2%)	4 (5.8%)	--
No	61 (96.8%)	65 (94.2%)	--
Family H/o CAD			
Yes	21 (33.3%)	35 (50.7%)	<0.05
No	42 (66.7%)	34 (49.3%)	--
Physical activity			
Inactive	32 (50.8%)	40 (58.0%)	--
Active	31 (49.2%)	29 (42.0%)	--
Fresh fruit consumption			
Yes	22 (34.9%)	29 (42.0%)	--
No	41 (65.1%)	40 (58.0%)	--
Smoker			
Yes	39 (61.9%)	24 (34.8%)	<0.05
No	24 (38.9%)	45 (65.2%)	--
Hypertension			
Yes	51 (81.0%)	44 (63.8%)	<0.05
No	12 (19.0%)	25 (36.2%)	--

BMI-Body mass index: MI-Myocardial infarction: CAD-Coronary artery dieases

Table 2. Distribution of patients studied in the two groups with reference to biochemical risk indices. Number of patients and their percentages are shown

Risk index	Non-Diabetic MI (n=63)	Diabetic MI (n=69)	p
HbA1c			
>7.5 g/dl	00 (0%)	29 (42.0%)	<0.000
≤7.5 g/dl	63 (100%)	40 (58.0%)	
TG/HDL-C ratio			
>3.5	24 (38.1%)	40 (58.0%)	<0.022
3.5 and <	39 (61.9%)	29 (42.0%)	
eGFR ml/min/1.73 m^2			
60 and >	28 (44.4%)	36 (52.2%)	0.37
<60	35 (55.6%)	33 (47.8%)	
Plasma MDA µM/L (Median=3.84)			
>3.84	26 (41.3%)	38 (55.0%)	0.11
3.84 and <	37 (58.7%)	31 (45.0%)	
Plasma FRAP µM/L (Median=514)			
>514	35 (55.6%)	29 (42.0%)	0.12
514 and <	28 (44.4%)	40 (58.0%)	
Localization of infarct			
Anterior	23 (36.5%)	39 (56.5%)	<0.002
Inferior	32 (50.8%)	15 (21.7%)	
Other	08 (12.7%)	15 (21.7%)	

HbA1c-Glycosylated Haemoglobin: TG-Tryglyceride: HDL-C- High density lipoprotein cholesterol: eGFR-estimated Glomerular filration rate: MDA- Melonal dialdehyde: FRAP-Ferric reducing ability of plasma

Table 3. Complications and outcome in course of MI among two studied groups shown in number and percentages in parenthesis

Complications/Course	Non-diabetic MI (n=63)	Diabetic MI (n=69)	p
Cardiogenic shock			
Yes	2 (3.2%)	3 (4.3%)	--
No	61 (96.8%)	66 (95.7%)	--
Ventricular fibrillation			
Yes	0 (0%)	6 (8.7%)	<0.05
No	63 (100%)	63 (91.3%)	--
Atrial fibrillation			
Yes	3 (4.8%)	6 (8.7%)	--
No	60 (95.2%)	63 (91.3%)	--
AV block			
Yes	2 (3.2%)	5 (7.2%)	--
No	61 (96.8%)	64 (92.8%)	--
Pulmonary edema			
Yes	3 (4.8%)	8 (11.6%)	--
No	60 (95.2%)	61 (88.4%)	--
Hospital stay (Days)			
>8	23 (36.5%)	42 (60.9%)	<0.005
8 and <	40 (63.5%)	27 (39.1%)	--
Death			
Yes	4 (6.3%)	6 (8.7%)	--
No	59 (93.7%)	63 (91.3%)	--

4. DISCUSSION

Most epidemiological investigations on cardiovascular diseases prevalent highly in developing countries are carried out in developed nations [16]. It is pertinent that relevant regional studies be conducted to appraise risk factors and their influence in ischaemic heart disease. Classic cardiovascular risk determinants as age, sex, smoking, unhealthy diet and physical inactivity, diabetes, hypertension and dyslipidaemia are widely analyzed. Some other predictors as lipoprotein A, apoB, homocysteine, High specificity C-reactive protein profiles are also studied. In present study acute myocardial infarction (MI) cases found bearing type 2 diabetes and those without it are compared for risk factor profiles.

Diabetes patients suffered MI significantly more frequently at relative younger age than non-diabetics. This is in agreement with enhanced aging and degenerative processes as atherosclerosis by diabetes. The proportion of female MI cases was significantly higher among diabetes group. Diabetic females are known to suffer higher cardiovascular morbidity than males [54]. Proportion of obese was significantly high among diabetic MI patients, as also reported by others [55]. Obesity is independent risk factor for cardiovascular morbidity and mortality [56,57]. Significantly higher instances of positive family history of ischaemic heart disease was observed in diabetes group, implying common significance of genotype/phenotype for diabetes and MI. Diabetics with such positive family history are however reported to have 7.6 times greater risk of suffering coronary disease than diabetics without family history [58]. Half the patients both in diabetes and non-diabetic group were found physically inactive and less than half in either group were adequately consuming fresh fruits. Significantly greater prevalence of smokers was detected among the non-diabetic group of MI patients. Relative risk of MI is well known to increase in tobacco smokers [59-61]. Smoking imposes oxidative stress that also may oxidize lipoprotein increasing their atherogenicity. Smoking also inhibits substance-P mediated tissue plasminogen activator release, increasing thrombotic risk especially in face of endothelial dysfunction [62]. Non-diabetic MI patients also had significantly greater prevalence of hypertension (81%) than among diabetics (65%). Hypertension is known to induce endothelial dysfunction, exacerbate atherosclerosis and increase plaque instability. The left ventricular hypertrophy, a usual consequence of hypertension leads to decreased coronary reserve, while increasing myocardial oxygen demand. Both mechanisms contribute to myocardial ischaemia [63].

Raised HbA1c is known to increase risk of ischaemic heart disease [64]. In particular, HbA1c levels above 7.5 g/dl, indicative of chronic hyperglycaemia are associated with diabetes complications [53]. Over 40% of diabetic MI patients had HbA1c level above 7.5g/dl and had poor glycaemic control as contributory factor. About 16% of normal weight individuals have been found to bear insulin resistance [65]. Insulin resistance is important contributor to risk of coronary disease [66,67]. The TG/HDL-C ratio is advocated indicator of insulin resistance [68,69], and best predicts cardiovascular risk [70]. There is a strong relationship between TG/HDL-C ratio and peak diameter of LDL-C [71]. TG brings significant change in LDL particle size, density, distribution and composition producing sdLDL.TG/HDL-C ratio >3.5 is predictor of presence of sdLDL phenotype with high sensitivity and specificity [72]. While lipid changes in diabetes occur due to increased free fatty acid flux, abnormal lipid profile in non-diabetics also associates increased risk of MI [73].

The proportion of MI cases with TG/HDL-C ratio >3.5 in the diabetics is significantly more 58% as compared to 38 % in non-diabetic group which is as per expectation [74]. A good proportion of dyslipidaemia even among non-diabetic MI patients may result from faulty dietary and lifestyle factors, smoking, renal insufficiency etc. 52% of diabetic and 44.4% of non-diabetic MI patients had eGFR values below 60 ml/kg/1.73 m^2 indicating decline in renal function, which is not significantly different. Decline in renal function is understood to cause graded increase in cardiovascular risk particularly in hypertensive and diabetic subjects [75,76]. Nephropathy directly correlates with coronary disease both in diabetics and non-diabetics [27,28]. Both the groups of MI patients had elevated profile of plasma malonaldehyde oxidative stress marker as well as depressed plasma antioxidant capacity without significant intergroup differences. Variety of risk factors may contribute to such state, viz. obesity, smoking, hyperglycaemia, hypertension, dyslipidaemia, renal dysfunction and poor consumption of antioxidant nutrients [19,50,74,77]. Oxidative stress and inflammation are significant risk factors for development of coronary artery disease [78]. Poor plasma antioxidant capacity contributes to greater severity of infarct [79,80].

Diabetes promotes atherogenesis and endothelial dysfunction and hence, more severe atherosclerosis involving multiple vessels [81]. Hyperinsulinaemia and hyperglycaemia inflict multi-vessel disease and disturbance in coagulation system including platelets and coagulation cascades toward worse scenario [82]. The diabetic subjects may have pre-existing subclinical cardiomyopathy [83]. Extensive coronary disease in diabetics may also cause regional left ventricular dysfunction [84] and excess neurohumoral activation [85]. There is also clustering of several cardiovascular risk factors in diabetics. The post MI course and complications in diabetic and non-diabetic patients shown in Table 3 reveals significant exclusive occurrence of ventricular arrhythmia among diabetics. Other cardiac sequel too and deaths were more apparently among diabetics but differences were not significant. Limited study size and tertiary centre level care may partly explain this. Very significantly however, the hospital stay was prolonged in diabetic MI cases. Alternately, the findings may indicate scope for successful mitigation of more severe aftermath of MI in diabetics.

In light of study findings, relevance of reported pharmacotherapeutic strategies for prevention and management of diabetic MI may be considered. Good control of postprandial hyperglycaemia as would reflect in HbA1c levels would be prudent to prevent ischaemic event [7,13,86]. Management of MI case in diabetics needs intensive control of hyperglycaemia below 180mg/dl with target of 140 to 90 mg/dl that would require insulin infusion [87]. Avoidance of hypoglycaemia is important at the same time as that worsens prognosis [88].

Because diabetes profoundly affects biology of cardiovascular disease, therapies particularly effective for diabetic MI and cardiac sequel deserve keen consideration. Peri-infarct pharmacotherapy would particularly need agents that improve endothelial function to promote collateral circulation for limiting infarct size amid extensive diabetic coronary disease. Angiotensin converting enzyme (ACE) inhibitors, statins and thiazolidinedione class of drugs are thus particularly appealing. ACE inhibitors may best counter neurohumoral activation and improve left ventricular failure and prevent ventricular arrhythmias [89]. They also facilitate bradykinin activity to enhance endothelial nitric oxide production.ACE inhibitors counter the increased activity of PAI-1 (plasminogen activator inhibitor-1) in diabetics and thus improve fibrinolysis [90]. ACE inhibitors improve insulin sensitivity [91] and

do not interfere with protective myocardial preconditioning following previous ischaemic attacks, unlike sulphonylurea drugs [92]. Beta-adrenergic blocking drugs reduce myocardial oxygen demand and shift source of energy production from fatty acids to glucose [93]. These would particularly benefit cases with sympathetic over activity caused by fatty acids and revealed in increased heart rate [94].

Indian subjects have high prevalence of atherogenic lipid profile with raised triglyceride and low HDL-C [95-97]. Dietary deficiency of polyunsaturated fats and physical inactivity may be contributory and thus deserve due address for correction. Supplementary drugs as statins, especially rosuvastatin as monotherapy or combination therapy of niacin/fibrates should be helpful. Increased oxidative stress promotes lipoprotein oxidation within vessel wall not amenable to correction by systemic metabolic control, and tissue antioxidant as α-lipoic acid supplement appears logical in diabetes management [98]. Post infarction secondary prevention has place for indefinite therapy with antiplatelet drug, beta blocker, statins and ACE inhibitor along with proper glycaemic and blood pressure control [99].

5. CONCLUSION

Study observations emphasize preventive role for dietary and lifestyle modification, weight reduction in diabetics and deterrence of smoking in non-diabetics as crucial. Management of hypertension is necessary measure while good glycaemic control and correction of dyslipidaemia is pertinent in diabetics to reduce incidence and possibly severity of MI relevant in central Indian context. ACE inhibitors, beta adrenergic blockers and statins besides emergent glycaemic control with insulin have particular therapeutic relevance in diabetic MI.

COMPETING INTERESTS

Author has declared that no competing interests exist.

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