SciMedCentral

Journal of Cardiology & Clinical Research

Research Article

Hypertension and Myocardial Infarction: A Study and Review

Abha Pandit*

Department of Medicine, Index Medical College, Hospital and Research Centre, India

***Corresponding author**

Abha Pandit, Department of Medicine, Index Medical College, Indore, Madhya Pradesh, India, Email: drabhaindore@gmail.com

Submitted: 18 July 2017

Accepted: 09 September 2017

Published: 11 September 2017

Keywords

- Hypertension
- Myocardial infarction
- Ischaemic heart disease
- HbA1c (Glycosylated hemoglobin

Abstract

Aim: Regional pattern and significance of risk determinants paving the way to incidence and fate of myocardial infarction (MI) in hypertensive subjects is attempted elaboration through comparison with contemporary cases of MI in normotensive patients in an 18 month long study at medical college hospital setting in central India.

Method: MI cases in 35 to 75 year age range without major unrelated systemic diseases were studied in hypertensive and normotensive categories. Demographic, clinical and laboratory information, as well as complications and outcome, were examined.

Results: MI in hypertension associated earlier age, more with positive family history of ischaemic heart disease, overweight, poor physical activity, deficient fresh fruit intake and the smoking habit. Uncontrolled diabetes significantly associated MI in hypertension as also deficient plasma antioxidant capacity. Hypertensive MI also had marginally increased rates of dyslipidaemia, and renal function declines. Incidence of various cardiac complications were insignificantly higher hospital stay was prolonged in hypertensive MI.

Conclusion: Pattern of risk factors and clinical course of MI in studied categories emphasized preventive dietary and physical activity measures for weight reduction, deterrence of smoking and good control of co-existing diabetes as most pertinent to bring down incidence and possibly severity of MI in hypertension, in central Indian context. Particular reference to antihypertensive cum cardioprotective drug classes angiotensin converting enzyme (ACE) inhibitors and angiotensin (AT)-II receptor blockers, beta blockers and calcium channel blockers is made in the context.

INTRODUCTION

Hypertension (HTN) is a major cause of mortality and morbidity and is expected to affect 1.56 billion people by 2025 [1].Currently 31.4% of Indian population is estimated to suffer HTN [2]. Elevated blood pressure is the key risk factor for cardiovascular disease [3,4]. Myocardial infarction (MI) is major cause of mortality and morbidity, often resulting as complication of HTN. Almost 40% of patients with ischaemic heart disease (IHD) who die suddenly have history of HTN [5]. Over 90% of MI victims bear many risk factors for coronary atherosclerosis besides HTN, viz. smoking habit, obesity, dyslipidaemia etc [6]. HTN is independent risk factor over other risk factors that may coexist [7]. Heart failure, stroke and other modes of cardiovascular mortality are higher among hypertensive patients [8,9]. Immediate and long term mortality after infarction is increased in hypertensives partly due to increased instances of myocardial and septal rupture [10].

Hypertension and IHD

HTN causes myocardial perfusion and ventricular function abnormalities, independent of the consequences associating ischaemic heart disease. HTN induces ventricular hypertrophy and together with that further compromises myocardial perfusion. The mechanisms include decrease in capillary density [11], failure of capacity to vasodilatation with increased muscle mass [12], an increased resistance for coronary collaterals [13] and more severe sub-endocardial involvement and larger ischaemic zones [13]. There is linear increase in risk of MI with increase of blood pressure status. HTN particularly raises the risk of MI in people under 65 years [14].

Commonest basis for MI is atherosclerotic disease of coronaries with thrombosis, spasm or plaque rupture, the precipitating entity being atheroma. HTN increases the prevalence of occlusive disease of coronaries [15]. Several studies have reported that hypertensive patients had higher instances of heart failure, stroke and cardiovascular death [9,16]. Hypertensive patients suffer multivessel coronary disease and complex coronary lesions contributing to worse outcome of MI. Aggravated atherosclerosis, left ventricular hypertrophy, insulin resistance, endothelial damage and ventricular arrhythmias all worsen prognosis [17]. Hypertensive MI patients suffer higher instances of postinfarct angina pectoris, recurrent silent

myocardial ischaemia and infarction, atrial and ventricular fibrillation, cardiogenic shock and pulmonary oedema than normotensive MI cases [9,18,19].

IHD risk due to HTN however is seen to vary among populations and is low in black Africans who generally display lower serum cholesterol profiles [20]. In contrast to western world HTN in Asians has unique traits in terms of response to antihypertensive medication, complications and outcome [21]. Due to high prevalence of HTN and diabetes, risk of stroke and coronary disease is high in Asia [22]. Significant regional variation in prevalence of coronary heart disease is documented in Asia [23,24]. Need for studies to asses nuances and features of hypertensive patients in Asia are emphasized [25], in order to optimally translate western recommendations of prevention and management. HTN related risk factors (including obesity and diabetes) and unhealthy lifestyle particularly, deserve keen evaluation [26]. The present observational study attempted appraisal of clinical profile and course of MI among normotensive and hypertensive cases in our specific region to generate evidence base for suitable clinical practices for prevention and management.

PATIENTS AND METHODS

The study is based on consecutively admitted MI patients in medical wards of Index Medical College, Indore (Central India), between January 2015 to June 2016 period. Patients above 35 years to 75 years age of either sex were included. Patients that had antecedent or subsequently diagnosed systemic diseases unrelated to occurrence of MI were excluded. Although records were not kept the excluded sample constituted around 10% of all screened. The exclusion was a preliminary measure to minimize complicated inferences on group differences. Of the 132 cases finally included, 37 were normotensive and the rest 95 were hypertensive, revealed either through history and/or detected upon examination (Blood pressure >140/90 mm Hg), at admission. MI diagnosis was based on patient meeting at least two of the following three criteria viz. typical chest pain/ discomfort lasting 30 minutes at least; an electrocardiographic prominent Q wave and/or ST segment elevation of >2mm in two contiguous precordial leads and abnormal rise of creatine kinase MB, indicative of myocardial necrosis.

The study protocol was approved by the college research ethical committee. For use of all information relating patient's condition and care, patient/attendants consent was obtained prior to inclusion, in proposed observational study. Intent and nature of study was made known and assurance of maintaining secrecy of identity was given.

Various risk factors of coronary disease, viz. age, sex, family history, physical activity profile, fresh fruit consumption, smoking habit, obesity (BMI>25kg/m^2), diabetes (HbA1c level >6.5g/ dl), dyslipidaemia (triglyceride (TG)/high density lipoprotein cholesterol (HDL-C) ratio >3.5), glomerular filtration rate (cut off 60ml/min/1.73m^2), plasma malonaldehyde level and plasma total antioxidant capacity were examined as earlier [27,28] and compared between the normotensive and hypertensive MI victims.

Family history of premature atherosclerosis was based on history of MI in parent or sibling male fewer than 55 and female less than 65 year age [29]. History of HTN, diabetes or cerebrovascular disorder constituted positive history irrespective of age. Body mass index (BMI) was calculated as ratio of body weight to height squared and >25kg/m^2 was taken as indicative of obesity in our patients [30]. Glomerular filtration rate was estimated from serum creatinine levels [31]. Physical activity status was assessed by enquiring usual activity and minimum 30 minute daily brisk exercise for at least 5 days in a week was deemed as active while less than that as inactive status. Fresh fruit intake was enquired as kind and quantity of fresh fruits consumed over the week and the month. An average minimum alternate day consumption of apple size fruit irrespective of economic status was taken as positive consumption and less as negative. Standard procedures were used for estimation of HbA1c, creatinine, lipid profile as previously described [28]. Oxidative stress markers and antioxidant capacity may vary as per time since occurance of event. These parameters were also estimated on the first day of admission. Plasma malonaldehyde level was determined as thiobarbituric acid reactive substances [32], as indicator of oxidative stress. Total plasma antioxidant capacity was determined by FRAP (ferric oxide reducing ability of plasma) method [33].Infarct localization and hospital outcome in respect to complications, length of hospital stay and mortality were also studied.

Relative frequency distributions of subjects in two compared groups in respect to every parameter were analyzed. Parameters employed cut off as yes/no, general median value or given normal value. Chi square test and when necessary (for small cell numbers), Fishers exact test statistic were used.

OBSERVATION AND RESULTS

Table 1, presents the comparison of presenting clinical and laboratory profile in two groups.

Higher proportion of hypertensive MI patients was younger under the median 62 years compared to the normotensive group but the gender composition was not different. Obesity defined by BMI above 25 was more prevalent among hypertensive MI group. Significantly higher prevalence of family history of coronary disease was present in hypertension group. History of past episodes of MI did not significantly differ although, more frequent in hypertension group. Significantly high proportion of the hypertensive victims reported physical inactivity in contrast to normotensives. Fresh fruit consumption was low and more so in hypertensive patients. Hypertensive MI group had significantly higher proportion of smokers.

As shown in table (2), prevalence of diabetes and dyslipidaemia was higher among hypertensive MI patients, although the difference was not statistically significant. Significantly high proportion of diabetic hypertyensives also exhibited HbA1c levels above 7.5g/dl indicating poor glycaemic control. Large proportions of MI cases in either group had reduced GFR under 60ml/min/1.73m^2. Median plasma malonaldehyde level of 3.84 μM/L seen in the MI patients is high, near double the normal 2 μM/L level. Plasma antioxidant activity indicated by median FRAP value of 514 μM/L is nearly halved 1000 μM/L normal level. Significantly higher proportion of hypertensive MI cases

Table 1: Distribution of patients in two studied groups around defined parameters.

Parameters	Normotensive MI (n=37)	%age	Hypertensive MI (n=95)	%age	p
Age					
>62 years	22	59.5	39	41	<0.057
≤62 years	15	40.5	56	59	--
Gender					
Male	31	83.8	67	70.5	<0.12
Female	6	16.2	28	29.5	--
BMI					
>25	15	40.5	56	59	<0.057
<25	22	59.5	39	41	--
Family history of CVD					
Present	21	56.8	35	36.8	<0.038
Absent	16	43.2	60	63.2	--
Past H/o CAD					
Present	1	2.7	7	7.4	--
Absent	36	97.3	88	92.6	--
Physical Activity					
Inactive	14	37.8	58	61	<0.016
Active	23	62.2	37	39	--
Fresh Fruit Consumption					
Yes	10	27	41	43.2	<0.087
No	27	73	54	56.8	--
Smoking Habit					
Yes	21	56.8	36	37.9	<0.049
No	16	43.2	59	62.1	--

Table 2: Distribution of patients in two studied groups around defined biochemical parameters.

Parameters	Normotensive MI (n=37)	%age	Hypertensive MI (n=95)	%age	p
Diabetes					
Present	15	40.5	54	56.8	<0.09
Absent	22	59.5	41	43.2	--
HbA1c (g/dl)					
>7.5	4	10.5	25	26.3	<0.053
<7.5	33	89.2	70	73.7	--
TG/HDL-C Ratio					
>3.5	15	40.5	49	51.6	--
<3.5	22	59.5	46	48.4	--
eGFR ml/min/1.73m^2					
>60	14	37.8	51	53.7	--
<60	23	62.2	44	46.3	--
Plasma MDA µM/L (Median=3.84)					
>3.84	15	40.5	50	52.6	--
<3.84	22	59.5	45	47.4	--
Plasma FRAP µM/L (Median=514)					
>514	24	64.9	42	44.2	<0.05
<514	13	35.1	53	55.8	--

display lower than median FRAP profile.

ECG based localization of infarct, the events in post MI clinical course and outcome of two groups were summarily examined. Hypertensive group had more of anterior infarcts while normotensives had more inferior infarct. Significantly high proportion of AMI cases of hypertensive group had prolonged hospital stay beyond median 8 days. However these values do not have adequate power and hence credence.

DISCUSSION

Classical cardiovascular risk factors as age, sex, smoking, unhealthy diet and physical inactivity, obesity, diabetes, dyslipidaemia and hypertension are widely studied for elucidation of population specific evidence base to support rational approaches to prevention and management. In the present context, the same were studied by dichotomy primarily based on blood pressure parameter in MI cases.

About 72% of all selected sample of MI were having hypertension. This reflects high incidence of hypertension in patients admitted with MI as also reported [34,35]. Many other studies found MI in hypertensives as occurring at relatively older age than in the normotensive people [36,37]. This may characterize as feature of local population with unexplored lifestyle and possibly other phenotypic determinants. Significantly more instances of positive family history of IHD in HTN group also imply significant shared genotype/phenotype of HTN and MI in studied patient population. Obesity and dyslipidaemia are also significantly more prevalent among hypertensive victims of MI. This may suggest large prevalence of metabolic syndrome among the hypertensives with MI. Overweight and obesity are independent coronary risk factors [38,39]. Poor consumption of fresh fruit would particularly detrimental in hypertensives depriving antioxidant nutrients. High prevalence of smoking among the HTN group would inflict oxidative stress increasing atherogenicity of lipids. Smoking is independent strong risk factor for MI [40,41].

Proportion of diabetics among the HTN group is marginally higher. In the group with HTN uncontrolled hyperglycaemia shown as plasma HbA1c above 7.5g/dl, was present in significantly high number there by constituting additional risk factor for IHD [42,43]. Basis for hypertension in majority of studied MI cases therefore could be metabolic syndrome. TG/HDL-C ratio >3.5 is indicative of insulin resistance, and atherogenic dyslipidaemia [44], which was similar in both. eGFR below 60ml/kg/1.73m^2 indicates renal insufficiency [28] which is a coronary disease risk factor [45,46], and was also marginally more prevalent among HTN group. Oxidative stress indicated by elevated plasma malonaldehyde levels was also similar in both groups. Plasma antioxidant capacity was low in both but significantly worse in HTN group. Oxidative stress and inflammation are significant CAD risk factors [47]. Poor plasma antioxidant capacity associates greater severity of infarct [48,49].

Higher incidence of variety of cardiac complications observed in hypertensive MI cases is in agreement with other reports [8,9]. Hypertensive cases have high likelihood of more severe atherosclerosis, endothelial damage, insulin resistance, ventricular hypertrophy and ventricular arrhythmias [50]. High likelihood of multiple vascular diseases in coronary arteries is known to occur in the hypertensives [51]. This may explain the observed significantly higher mortality of AMI in hypertensive group. Hypertension is associated with increased risk of atrial fibrillation [52]. High rate of adverse outcome in AMI in hypertension including left ventricular failure and recurrent AMI are reported [53].

Hypertension accelerates atherogenesis primarily dependent on cholesterol. Coronary artery atherosclerotic disease is 2 to 3 fold more prevalent among hypertensive individuals in contrast to that in normotensive people [54]. Clustering of cardiovascular risk factors causes high rate of cardiovascular events [55], and this is frequently the case in Indian patients [56]. Risk increases with increase in number of risk factors [57]. Reduction in number of risk factors yields healthier life and longevity [58,59]. The in hospital mortality in MI cases varies between 7.7% to 19.2% across the globe, while mortality over one year post infarct touches 23% to 25.3% [60,61]. Promotion of healthy diet and increase in physical activity to maintain optimal weight ought to be very intensively persued as cardinal preventive measures.

Increased prevalence of decline in eGFR is found in hypertensive MI cases and there is frequent altered blood sugar stress response associating acute high insulin resistance [62]. Careful metabolic monitoring is therefore required in hypertensive MI cases [63]. Aggressive blood pressure reduction may be pertinent in cases with coronary disease. Control of blood pressure is often suboptimal in clinical practice and this may be the case in over 50% of cases as per Asian study [64]. Association of physicians of India ordinarily encourages blood pressure control at 140/80 mm Hg [65]. High risk cases with clustered risk factors would need tighter control while very old would be better served with systolic 150mm Hg. Optimal treatment of hypertension frequently would require combination drug therapy for prevention of cardiovascular events [66].

REFERENCES

1. Angeli F,Reboldi G,Verdecchia P.The 2014 hypertension guidelines: implications for patients and practitioners in Asia. Heart Asia. 2015; 7: 21-25.
2. Neupane D, McLachlan CS, Sharma R, Gyawali B, Khanal V, Mishra SR, et al. Prevalence of hypertension in member countries of South Asian Association for Regional Cooperation (SAARC): systematic review and meta-analysis. Medicine (Baltimore). 2014; 93: e74.
3. Woodward M, Barzi F, Martiniuk A, Fang X, Gu DF, Imai Y, et al. Asia Pacific Cohort Studies Collaboration. Cohort profile: the Asia Pacific Cohort Studies Collaboration. Int J Epidemiol. 2006; 35: 1412-1416.
4. Lawes CM, Rodgers A, Bennett DA, Parag V, Suh I, Ueshima H, MacMahon S. Asia Pacific Cohort Studies Collaboration. Blood pressure and cardiovascular disease in the Asia Pacific region. J Hypertens. 2003; 21:707-716.
5. Law M,Wald N,Morris J. Lowering blood pressure to prevent myocardial infarction and stroke:a new preventive strategy.Health Technol Assess 2003;7:1-94
6. Jefferson BK Topol EJ. Molecular mechanisms of myocardial infarction. Curr Probl Cardiol. 2005; 30: 333-374.
7. Kannel WB, Gordon T, Schwartz MJ. Systolic versus diastolic blood pressure and risk of coronary heart disease. The Framingham studyAm J Cardiol. 1971; 27: 335-346.
8. Richards AM , Nicholls MG, Troughton RW, Lainchbury JG, Elliott J, Frampton C et al. Antecedant hypertension and heart failure after myocardial infarction. J Am Coll Cardiol. 2002; 39: 1182-1188.
9. Thune JJ, Signorovitch J, Kober L, Velazquez EJ, McMurray JJ, Califf RM et al. Effect of antecedent hypertension and follow-up blood pressure on outcomes after high-risk myocardial infarction. Hypertension. 2008; 51: 48-54.
10. Cruz H, Cruz JC, Badui E, Galindo ME, Solorio S, Bojorges R. Cardiac rupture in acute myocardial infarct. Presentation of 20 postmortem cases. Arch Inst Cardiol Mex. 1997; 67: 51-58.
11. Rakusan K, Flanagan MF, Geva T, Southern J, Van Praagh R. Morphometry of human coronary capillaries during normal growth and the effect of age in left ventricular pressure-overload hypertrophy. Circulation. 1992; 86: 38-46.
12. Mueller TM, Marcus ML, Kerber RE, Young JA, Barnes RW, Abboud FM. Effect of renal hypertension and left ventricular hypertrophy on the coronary circulation in dogs. Circ Res. 1978; 42: 543-549.

13. Mueller TM, Tomanek RJ, Kerber RE, Marcus ML. Myocardial infarction in dogs with chronic hypertension and left ventricular hypertrophy. Am J Physiol. 1980; 239: 731-735.

14. Ali I, Akman D, Bruun NE, Køber L, Brendorp B, Ottesen M, Møller J, Torp-Pedersen C. Importance of a history of hypertension for the prognosis after acute myocardial infarction--for the Bucindolol Evaluation in Acute myocardial infarction Trial (BEAT) study group. Clin Cardiol. 2004; 27: 265-269.

15. Robertson WB, Strong JP. Atherosclerosis in persons with hypertension and diabetes mellitus. Lab Invest. 1968; 18: 538-551.

16. Richards AM, Nicholls MG, Troughton RW, Lainchbury JG, Elliott J, Frampton C et al. Antecedent hypertension and heart failure after myocardial infarction. J Am Coll Cardiol. 2002; 39: 1182-1188.

17. Pedrinelli R, Ballo P, Fiorentini C, Denti S, Galderisi M, Ganau A, et al. Hypertension and acute myocardial infarction: an overview. J Cardiovasc Med (Hagerstown). 2012; 13:194-202.

18. Wang Y, Zheng Y, Zhang W, Yu H, Lou K, Zhang Y et al. Polymorphisms of KDR gene are associated with coronary heart disease. J Am Coll Cardiol. 2007; 50: 760-767.

19. Rembek M, Goch A, Goch J. The clinical course of acute ST-elevation myocardial infarction in patients with hypertension. Kardiol Pol. 2010; 68: 157-163.

20. Dominguez LJ, Galioto A, Pineo A, Ferlisi A, Vernuccio L, Belvedere M, et al. Blood pressure and cardiovascular risk profiles of Africans who migrate to western country. Ethn Dis. 2008; 18: 512-518.

21. Kearney PM, Whelton M, Reynolds K, Whelton PK, He J. Worldwide prevalence of hypertension: a systematic review. J Hypertens. 2004; 22: 11-19.

22. Woodward M, Huxley H, Lam TH, Barzi F, Lawes CM, Ueshima H; Asia Pacific Cohort Studies Collaboration.. A comparison of the associations between risk factors and cardiovascular disease in Asia and Australasia. Eur J Cardiovasc Prev Rehabil. 2005; 12: 484-91.

23. Nag T, Ghosh A. Cardiovascular disease risk factors in Asian Indian population: A systematic review. J Cardiovasc Dis Res. 2013; 4: 222-228.

24. Wang JG, Li Y. Characteristics of hypertension in the Chinese population. Curr Hypertens Rep. 2012; 14: 410-415.

25. Chung N, Baek S, Chen MF, Liau CS, Park CG, Park J, et al. Expert recommendations on the challenges of hypertension in Asia. Int J Clin Pract. 2008; 62: 1306-1312.

26. Singh RB, Suh IL, Singh VP, Chaithiraphan S, Laothavorn P, Sy RG, et al. Hypertension and stroke in Asia: prevalence, control and strategies in developing countries for prevention. J Hum Hypertens. 2000; 14: 749-763.

27. Pandit A, Pandey AK. Liver dysfunction in pulmonary tuberculosis patients on DOTS:a study and review.Journal of Gastroenterology and Hepatology Research. 2016; 5: 2254-2260.

28. Pandit A, Pandey AK. Estimated glomerular filtration rate and associated clinical and biochemical charecteristics in type 2 diabetes patients. Advances in Diabetes and Metabolism. 2016; 4: 65-72.

29. Nasir K, Michos ED, Rumberger JA, Braunstein JB, Post WS, Budoff MJ, Blumenthal RS. Coronary artery calcification and family history of premature coronary heart disease: sibling history is more strongly associated than parental history. Circulation. 2004; 110: 2150-2156.

30. Snehalatha C, Viswanathan V, Ramachandran A. Cutoff values for normal anthropometric variables in asian Indian adults. Diabetes Care. 2003; 26:1380-1384.

31. Leavy AS, Greene T, Kusek JW, Beck GJ. A simplified equation to predict glomerular filtration from serum creatinine.J Am Soc Nephrol. 2000; 11: 0828.

32. Sangeetha P, Das UN, Koratkar R, Suryaprabha P. Increase in free radical generation and lipid peroxidation following chemotherapy in patients with cancer. Free Radic Biol Med. 1990; 8: 15-19.

33. Kamimori H, Hamashima Y, Konishi M. Determination of carnitine and saturated-acyl group carnitines in human urine by high-performance liquid chromatography with fluorescence detection. Anal Biochem. 1994; 218: 417-424.

34. Frazier CG, Shah SH, Armstrong PW, Bhapkar MV, McGuire DK, Sadowski Zet al Prevalence and management of hypertension in acute coronary syndrome patients varies by sex: observations from the Sibrafiban versus aspirin to Yield Maximum Protection from ischemic Heart events postacute cOroNary sYndromes (SYMPHONY) randomized clinical trials. Am Heart J. 2005; 150: 1260-1267.

35. Majahalme SK, Smith DE, Cooper JV, Kline-Rogers E, Mehta RH, Eagle KA, Bisognano JD. Comparison of patients with acute coronary syndrome with and without systemic hypertension. Am J Cardiol. 2003; 92: 258-263.

36. Bertomeu V, Cabades A, Morillas P, Cebrian J, Colomina F, Valencia Jet al. Clinical course of acute myocardial infarction in the hypertensive patient in Eastern Spain: the PRIMVAC registry. Heart Lung. 2006; 35:206-211.

37. Gustafsson F, Køber L, Torp-Pedersen C, Hildebrandt P, Ottesen MM, Sonne B, et al. Long term prognosis after acute myocardial infarction in patients with history of arterial hypertension. TRACE study group. Eur Heart J. 1998; 19: 588-594.

38. Prospective Studies Collaboration, Whitlock G, Lewington S, Sherliker P, Clarke R, Emberson J, et al. Body-mass index and cause-specific mortality in 900 000 adults: collaborative analyses of 57 prospective studies. Lancet. 2009; 373:1083-1096.

39. Wilson PW, D'Agostino RB, Sullivan L, Parise H, Kannel WB. Overweight and obesity as determinants of cardiovascular risk: the Framingham experience. Arch Intern Med. 2002; 162: 1867-1872.

40. Prescott E, Hippe M, Schnohr P, Hein HO, Vestbo J. Smoking and risk of myocardial infarction in women and men: longitudinal population study. BMJ. 1998; 316: 1043-1047.

41. Pais P, Pogue J, Gerstein H, Zachariah E, Savitha D, Jayprakash S, et al. Risk factors for acute myocardial infarction in Indians: a case-control study. Lancet. 1996; 348: 358-363.

42. Garg N, Moorthy N, Kapoor A, Tewari S, Kumar S, Sinha A, et al. Hemoglobin A(1c) in nondiabetic patients: an independent predictor of coronary artery disease and its severity. Mayo Clin Proc. 2014; 89: 908-916.

43. Gillett MJ. International Expert Committee report on the role of the A1c assay in the diagnosis of diabetes: Diabetes Care 2009; 32: 1327-1334. Clin Biochem Rev. 2009; 30:197-200.

44. McLaughlin T, Reaven G, Abbasi F, Lamendola C, Saad M, Waters D, et al. Is there a simple way to identify insulin-resistant individuals at increased risk of cardiovascular disease? Am J Cardiol. 2005; 96: 399-404.

45. Rocco MV, Yan G, Gassman J, Lewis JB, Ornt D, Weiss B, et al. Comparison of causes of death using HEMO Study and HCFA end-stage renal disease death notification classification systems. The National Institutes of Health-funded Hemodialysis. Health Care Financing Administration. Am J Kidney Dis. 2002; 39: 146-153.

46. Deveci OS, Kabakci G, Tulumen E, Okutucu S, Aksoy H, Kaya EB, et al The relationship between microalbuminuria and the presence and

extent of coronary atherosclerosis. Angiology. 2010; 61: 184-191.

47. Kostner K, Hornykewycz S, Yang P, Neunteufl T, Glogar D, Weidinger F, et al. Is oxidative stress causally linked to unstable angina pectoris? A study in 100 CAD patients and matched controls. Cardiovasc Res. 1997; 36: 330-336.

48. Fazendas P, João IF, Llobet S, Matias F, Pereira H, Oliveira LM, Carrageta M. [Plasma total anti-oxidant status in young survivors of myocardial infarction]. [Article in Portuguese] Rev Port Cardiol. 2000; 19: 463-467.

49. Yegin A, Yegin H, Alicigüzel Y, Deger N, Semiz E. Erythrocyte selenium-glutathione peroxidase activity is lower in patients with coronary atherosclerosis. Jpn Heart J. 1997; 38: 793-798.

50. Yurenev AP, DeQuattro V, Devereux RB. Hypertensive heart disease: relationship of silent ischemia to coronary artery disease and left ventricular hypertrophy. Am Heart J. 1990; 120: 928-933.

51. Eftekhari A, Mathiassen ON, Buus NH, Gotzsche O, Mulvany MJ, Christensen KL. Disproportionally impaired microvascular structure in essential hypertension. J Hypertens. 2011; 29: 896-905.

52. Abrignani MG, Dominguez LJ, Biondo G, Di Girolamo A, Novo G, Barbagallo M, et al. In-hospital complications of acute myocardial infarction in hypertensive subjects. Am J Hypertens. 2005; 18: 165-170.

53. Rabkin SW, Mathewson FA, Tate RB. Prognosis after acute myocardial infarction: relation to blood pressure values before infarction in a prospective cardiovascular study. Am J Cardiol. 1977; 40: 604-610.

54. Hambrecht R, Wolf A, Gielen S, Linke A, Hofer J, Erbs S, Schoene N, Schuler G. Effect of exercise on coronary endothelial function in patients with coronary artery disease. N Engl J Med. 2000; 342: 454-460.

55. Malik S, Wong ND, Franklin SS, Kamath TV, L'Italien GJ, Pio JR, Williams GR. Impact of the metabolic syndrome on mortality from coronary heart disease, cardiovascular disease, and all causes in United States adults. Circulation. 2004; 110: 1245-1250.

56. Gupta S, Gudapati R, Gaurav K, Bhise M. Emerging risk factors for cardiovascular diseases: Indian context. Indian J Endocrinol Metab. 2013; 17: 806-814.

57. Ji J, Pan E, Li J, Chen J, Cao J, Sun D et al. Classical risk factors of cardiovascular disease among Chinese male steel workers: a prospective cohort study for 20 years. BMC Public Health. 2011; 11: 497.

58. Stamler J, Stamler R, Neaton JD, Wentworth D, Daviglus ML, Garside D et al. Low risk-factor profile and long-term cardiovascular and noncardiovascular mortality and life expectancy: findings for 5 large cohorts of young adult and middle-aged men and women. JAMA. 1999; 282: 2012-2018.

59. Daviglus ML, Liu K, Pirzada A, Yan LL, Garside DB, Feinglass Jet al. Favorable cardiovascular risk profile in middle age and health-related quality of life in older age. Arch Intern Med. 2003; 163: 2460-2468.

60. Ahmadi A, Mobasheri M, Hashemi-Nazari SS, Baradaran A, Choobini ZM. Prevalence of hypertension and type 2 diabetes mellitus in patients with colorectal cancer and their median survival time: A cohort study. J Res Med Sci. 2014; 19: 850-854.

61. Chung SC, Gedeborg R, Nicholas O James S, Jeppsson A, Wolfe C, et al. Acute myocardial infarction: a comparison of short-term survival in national outcome registries in Sweden and the UKLancet. 2014; 383: 1305-1312.

62. Lazzeri C, Valente S, Chiostri M, Attanà P, Picariello C, Gensini GF. Impact of hypertension on short- and long-term prognoses in patients with ST elevation myocardial infarction and without previously known diabetes. Heart Vessels. 2012; 27: 370-376.

63. Reinstadler SJ, Stiermaier T, Eitel C, Saad M, Metzler B, de Waha S et al. Antecedent hypertension and myocardial injury in patients with reperfused ST-elevation myocardial infarction. J Cardiovasc Magn Reson. 2016; 18: 80.

64. Park JB, Kario K, Wang JG. Systolic hypertension: an increasing clinical challenge in Asia. Hypertens Res. 2015; 38: 227-236.

65. Association of Physicians of India. Indian guidelines on hypertension (I.G.H.) - III. 2013. J Assoc Physicians India. 2013; 61: 6-36.

66. Matsuzaki M, Ogihara T, Umemoto S, Rakugi H, Matsuoka H, Shimada KA, et al. Combination Therapy of Hypertension to Prevent Cardiovascular Events Trial Group. Prevention of cardiovascular events with calcium channel blocker-based combination therapies in patients with hypertension: a randomized controlled trial. J Hypertens. 2011; 29: 1649-1659.

Cite this article

Pandit A (2017) Hypertension and Myocardial Infarction: A Study and Review. J Cardiol Clin Res 5(6): 1118.