

Original research article

A Study focusing Emerging Risk Factors in Patients of Acute Myocardial Infarction in Bihar

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Abstract

Aim: To determine the risk factors associated with patients of acute myocardial infarction.

Materials & Methods: This study was a nested case-control study was conducted in the Department of Medicine, Narayan Medical College & Hospital Sasaram, Bihar. The patients were selected from the OPD/in-hospital patients. A total of 1300 patients who fulfilled the inclusion criteria were included in the study. Study participants were randomly allocated to two groups: cases (n=650) & control (n=650) groups respectively. The study duration was one year.

Results: Case patients had a mean (SD) age of 65.4 (11.2) years and control individuals had a mean (SD) age of 65.7 (11.8) years. Compared with controls, in-hospital AMI cases were significantly more likely to occur in intensive care unit settings (24.8% vs. 8.2%) and to have a history of atherosclerotic disease (i.e., myocardial infarction, percutaneous coronary intervention, coronary artery bypass graft, cerebrovascular disease, or peripheral vascular disease). Coronary disease risk factors of hypertension, hyperlipidemia, and diabetes were also more common in patients with AMI.

Conclusion: From case - control design, factors associated with an increased risk of in-hospital AMI included history of atherosclerosis, traditional atherosclerotic risk factors, and markers of physiological stress. Additional research to define risk reduction and optimal treatment strategies of in-hospital AMI are needed to address this common and high-risk condition.

Keywords: risk factors, acute myocardial infarction

Introduction

Patients who survive an acute myocardial infarction (AMI) are at high risk of major cardiovascular events following discharge, although risk may vary considerably between patients. Lifestyle changes, quality of care, and treatment with guideline-directed medical therapy after AMI are all important for secondary prevention. Patient characteristics are also associated with risk of cardiovascular events.

Identification of these characteristics would better inform patients, families, and physicians about the risk of cardiovascular events following discharge and help ensure intensive follow-up and risk factor modification. Efforts to identify risk factors have focused on the period immediately after initial hospitalization for AMI and have not provided a longer-term perspective. Moreover, studies on long-term outcomes after an AMI predominately focus on mortality. [1-4]

Moreover, coronary artery disease tends to emerge earlier in life, and thus mortality rate ratios are greatest in the youngest South Asians compared with other ethnic groups. [5, 6] However, acute myocardial infarction in young South Asians has not been extensively studied, and most of the existing data are on migrant South Asian populations. To determine the risk factors associated with patients of acute myocardial infarction.

Materials & Methods:

This study was a nested case-control and matched cohort study of cases and controls to determine risk factors associated with AMI conducted in the Department of Medicine, Narayan Medical College & Hospital, Sasaram, Bihar. The patients were selected from the OPD/in-hospital patients. The study duration was one year.

Inclusion Criteria:

Patients in the control group included patients with in-hospital AMI who were aged 50 years or older at the time of the event and admitted to the hospital. The control group included patients aged 50 years or older who were admitted to a medical bed section with a diagnosis other than ischemic heart disease (*ICD-9* diagnosis codes 410-414) and did not receive a diagnosis of AMI at any time during their hospitalization.

Exclusion Criteria:

Patients with AMI onset within 24 hours of admission were excluded to ensure cases represented AMIs that truly occurred in-hospital and patients who were transferred to another care facility.

A total of 1300 patients who fulfilled the inclusion criteria were included in the study. Study participants were randomly allocated to two groups: cases (n=650) & control (n=650) groups respectively.

All the data were tabulated and entered in Microsoft Excel Sheet. Statistical analysis was carried out using SPSS version 21.

Results:

Table 1. Case patients had a mean (SD) age of 65.4 (11.2) years and control individuals had a mean (SD) age of 65.7 (11.8) years. Compared with controls, in-hospital AMI cases were significantly more likely to occur in intensive care unit settings (24.8% vs. 8.2%) and to have a history of atherosclerotic disease (i.e., myocardial infarction, percutaneous coronary intervention, coronary artery bypass graft, cerebrovascular disease, or peripheral vascular disease). Coronary disease risk factors of hypertension, hyperlipidemia, and diabetes were also more common in patients with AMI.

Table 2: In case-control studies, the calculated exposure odds ratio approximates the disease odds ratio when AMI is sufficiently rare. From the final model, variables associated with an increased risk of in-hospital AMI included being married, a history of coronary artery disease, prior myocardial infarction, peripheral vascular disease, elevated heart rate,

Candidate variables that were associated with a lower risk of in-hospital AMI included low heart rate, atrial fibrillation, history of anemia, and depression.

Table 1: Patient Characteristics on Admission and In-Hospital Variables Prior to Event for Acute Myocardial Infarction Cases and Controls:

Characteristics	Total	%	Cases	%	Controls	%	p-value
	N=1300		N=650		N=650		
Age, mean (SD), y	65.2	11.3	65.4	11.2	65.7	11.8	0.7
Married	1080	83.08	564	86.76	516	79.38	0.05
Comorbidities and risk factors							
Hypertension	758	58.31	401	61.69	357	54.92	0.03
Hyperlipidemia	690	53.08	372	57.23	318	48.92	0.03
Tobacco use	300	23.08	163	25.08	137	21.08	0.001
Coronary artery disease	894	68.77	462	71.08	432	66.46	0.001
Prior myocardial infarction	215	16.54	117	16.54	98	16.54	0.05
Prior percutaneous coronary intervention	173	13.31	113	13.31	60	13.31	0.05
Prior coronary artery bypass graft	290	22.31	182	22.31	108	22.31	0.001
Heart failure	395	30.38	192	30.38	192	30.38	0.6
Cerebrovascular disease	362	27.85	49	27.85	49	27.85	0.001
Peripheral vascular disease	317	24.38	189	29.08	128	19.69	0.001
Atrial fibrillation	297	22.85	165	25.38	132	20.31	0.62
Diabetes	733	56.38	456	70.15	277	42.62	0.81
Chronic obstructive pulmonary disease	541	41.62	387	59.54	154	23.69	0.94
Obstructive sleep apnea	152	11.69	80	12.31	72	11.08	0.21
Chronic kidney disease	452	34.77	257	39.54	195	30	0.001
Liver disease	178	13.69	106	16.31	72	11.1	0.21
Anemia	391	30.08	184	28.31	207	31.8	0.24
Depression	385	29.62	174	26.77	211	32.5	0.3
Alcohol dependence or abuse	271	20.85	99	15.23	172	26.5	0.36
Posttraumatic stress disorder	113	8.692	60	9.231	53	8.15	0.27
Dementia	289	22.23	113	17.38	176	27.1	0.01
Malignant neoplasm	579	44.54	252	38.77	327	50.3	0.17
Coagulopathy	10	0.769	6	0.923	4	0.62	0.001
Fluid or electrolyte disorder	51	3.923	24	3.692	27	4.15	0.55
Gastrointestinal bleed	130	10	69	10.62	61	9.38	0.36
Presenting characteristics							
Body mass index							
Underweight (<18.5)	159	12.23	82	12.62	77	11.85	0.60
Normal (18.5 to <25)	547	42.08	288	44.31	259	39.85	
Overweight (25 to <30)	374	28.77	152	23.38	222	34.15	
Obese (≥30)	220	16.92	110	16.92	110	16.92	
Heart rate, beats/min							
Low (<60)	156	12	66	10.15	90	13.85	0.01
Normal (60-100)	842	64.77	303	46.62	539	82.92	
High (>100)	302	23.23	183	28.15	119	18.31	
Systolic blood pressure, mm Hg							
Low (<90)	68	5.231	43	6.615	25	3.846	0.5
Normal (90-120)	531	40.85	259	39.85	272	41.85	
Borderline (121-139)	357	27.46	180	27.69	177	27.23	
High (>139)	247	19	126	19.38	121	18.62	
Hypoxia (<90%)	97	7.462	63	9.692	34	5.231	0.01

Table 2: Independent Risk Factors Associated With In-Hospital Acute Myocardial Infarction

Variables	Odds ratio (95% CI)	p-value
Married	1.8 (1.3-2.4)	0.001
History of anemia	0.5 (0.3-0.7)	0.001
Coronary artery disease	1.9 (1.4-2.7)	0.001
Depression	0.7 (0.5-0.9)	0.35
Prior myocardial infarction	2.0 (1.3-3.1)	0.001
Peripheral vascular disease	2.0 (1.4-3.0)	0.001
Heart rate, beats/min		
Low vs. normal	0.5 (0.3-0.9)	0.001
High vs. normal	3.6 (2.5-5.3)	

Discussion:

Risk factors associated with an increased risk of in-hospital AMI included a history of atherosclerotic disease and cardiovascular risk factors in addition to markers of physiological stress (e.g., elevated heart rate, low hemoglobin, and elevated white blood cell count). While contemporary studies suggest the mortality for AMI that begins outside the hospital is approximately 13% at 30 days^[7] and 25% at 1 year.^[8]

The occurrence of major cardiovascular events following discharge is unobservable at discharge and associated with unknown factors after discharge. Such characteristics present a challenge for predictive model development based on conventional regression methods. Latent class analysis is increasingly being used in outcomes research^[9-14] and can stratify patients into subgroups based on their risk factors at discharge when outcome information is absent in a training sample.

There are controversial data on whether consanguineous marriages lead to a higher incidence of congenital and acquired diseases in the offspring. Our study reports a relation between parental consanguinity and early myocardial infarction, independent of family history of cardiovascular disease. It is reasonable to speculate that consanguinity increases the likelihood of inheriting genetic factors—identified as well as unidentified—leading to premature ischemic heart disease.^[15, 16]

Patients surviving AMI have a higher risk of re-infarction and cardiovascular mortality compared with stable coronary artery disease patients^[17, 18]. Dutta et al.^[19] demonstrated that in Apoe^{-/-} mice, after coronary artery ligation, the size of aortic plaques increased and vulnerable lesion morphology was induced with higher inflammatory cell content and protease activity, fuelled by persistently increased myeloid cell flux to atherosclerotic sites activated by heightened sympathetic nervous system activity. Recent studies^[20, 21] also demonstrated in humans that F-FDG uptake increased in infarcted myocardium and it was correlated with uptake of remote myocardium, spleen and bone marrow. Besides, a correlation was found between spleen uptake and carotid artery uptake.

Several studies attempting to explain these findings demonstrated that AMI patients have multiple vulnerable plaques that can lead to future cardiovascular events^[22-24]. However, AMI itself clearly accelerates atherosclerosis by infarct-triggering burst of systemic inflammation aimed at repair of injured heart.

Conclusion:

From case - control design, factors associated with an increased risk of in-hospital AMI included history of atherosclerosis, traditional atherosclerotic risk factors, and markers of physiological stress. Additional research to define risk reduction and optimal treatment strategies of in-hospital AMI are needed to address this common and high-risk condition.

References:

1. Tu JV, Austin PC, Walld R, Roos L, Agras J, McDonald KM. Development and validation of the Ontario acute myocardial infarction mortality prediction rules. *J Am Coll Cardiol*. 2001;37(4):992-997.
2. Smolderen KG, Buchanan DM, Gosch K, et al. Depression treatment and 1-year mortality following acute myocardial infarction: insights from the TRIUMPH registry (Translational Research Investigating Underlying Disparities in Acute Myocardial Infarction Patients' Health Status). *Circulation*. 2017;135(18):1681-1689.
3. Plakht Y, Shiyovich A, Gilutz H. Predictors of long-term (10-year) mortality postmyocardial infarction: age-related differences. Soroka Acute Myocardial Infarction (SAMI) Project. *J Cardiol*. 2015;65(3):216-223.
4. Ketchum ES, Dickstein K, Kjekshus J, et al. The Seattle Post Myocardial Infarction Model (SPIM): prediction of mortality after acute myocardial infarction with left ventricular dysfunction. *Eur Heart J Acute Cardiovasc Care*. 2014;3(1):46-55.
5. Reddy KS, Yusuf S. Emerging epidemic of cardiovascular disease in developing countries. *Circulation* 1998;97:596-601.
6. Anand SS, Yusuf S, Vuksan V, et al. Differences in risk factors, atherosclerosis, and cardiovascular disease between ethnic groups in Canada: the study of health assessment and risk in ethnic groups (SHARE). *Lancet* 2000;356:279-84.
7. Wadhwa RK, Joynt Maddox KE, Wang Y, Shen C, Bhatt DL, Yeh RW. Association between 30-day episode payments and acute myocardial infarction outcomes among Medicare beneficiaries. *Circ Cardiovasc Qual Outcomes*. 2018;11(3):e004397.
8. Spatz ES, Beckman AL, Wang Y, Desai NR, Krumholz HM. Geographic variation in trends and disparities in acute myocardial infarction hospitalization and mortality by income levels, 1999-2013. *JAMA Cardiol*. 2016;1(3):255-265.
9. Kim M, Wall MM, Li G. Applying latent class analysis to risk stratification for perioperative mortality in patients undergoing intraabdominal general surgery. *Anesth Analg*. 2016;123(1):193-205.
10. Miller CR, Sabagh G, Dingman HF. Latent class analysis and differential mortality. *J Am Stat Assoc*. 1962;57 (298):430-438.
11. Crow SJ, Swanson SA, Peterson CB, Crosby RD, Wonderlich SA, Mitchell JE. Latent class analysis of eating disorders: relationship to mortality. *J Abnorm Psychol*. 2012;121(1):225-231.
12. Leigh L, Hudson IL, Byles JE. Sleeping difficulty, disease and mortality in older women: a latent class analysis and distal survival analysis. *J Sleep Res*. 2015;24(6):648-657.
13. Evenson KR, Herring AH, Wen F. Accelerometry-assessed latent class patterns of physical activity and sedentary behavior with mortality. *Am J Prev Med*. 2017;52(2):135-143.
14. Ferrat E, Audureau E, Paillaud E, et al; ELCAPA Study Group. Four distinct health profiles in older patients with cancer: latent class analysis of the prospective ELCAPA cohort. *J Gerontol A Biol Sci Med Sci*. 2016;71(12): 1653-1660.
15. Ulusoy M, Tuncbilek E. Consanguineous marriage in Turkey and its effects on infant mortality. *Nufusbil Derg* 1987;9:7-26.
16. Modell B, Darr A. Science and society: genetic counselling and customary consanguineous marriage. *Nat Rev Genet* 2002;3:225-9.

17. Goldstein JA, Demetriou D, Grines CL, Pica M, Shoukfeh M, O'Neill WW. Multiple complex coronary plaques in patients with acute myocardial infarction. *The New England journal of medicine*. 2000; 343 (13):915–22. Epub 2000/09/28. doi:
18. Milonas C, Jernberg T, Lindbäck J, Agewall S, Wallentin L, Stenestrand U. Effect of Angiotensin-converting enzyme inhibition on one-year mortality and frequency of repeat acute myocardial infarction in patients with acute myocardial infarction. *The American journal of cardiology*. 2010; 105(9):1229–34. Epub 2010/04/21.
19. Dutta P, Courties G, Wei Y, Leuschner F, Gorbатов R, Robbins CS, et al. Myocardial infarction accelerates atherosclerosis. *Nature*. 2012; 487(7407):325–9. Epub 2012/07/06.
20. Kim EJ, Kim S, Kang DO, Seo HS. Metabolic activity of the spleen and bone marrow in patients with acute myocardial infarction evaluated by 18f-fluorodeoxyglucose positron emission tomographic imaging. *Circulation Cardiovascular imaging*. 2014; 7(3):454–60. Epub 2014/02/04.
21. Wollenweber T, Roentgen P, Schäfer A, Schatka I, Zwadlo C, Brunkhorst T, et al. Characterizing the inflammatory tissue response to acute myocardial infarction by clinical multimodality noninvasive imaging. *Circulation Cardiovascular imaging*. 2014; 7(5):811–8. Epub 2014/07/23.
22. Rioufol G, Finet G, Ginon I, André-Fouët X, Rossi R, Vialle E, et al. Multiple atherosclerotic plaque rupture in acute coronary syndrome: a three-vessel intravascular ultrasound study. *Circulation*. 2002; 106 (7):804–8. Epub 2002/08/15.
23. Asakura M, Ueda Y, Yamaguchi O, Adachi T, Hirayama A, Hori M, et al. Extensive development of vulnerable plaques as a pan-coronary process in patients with myocardial infarction: an angioscopic study. *Journal of the American College of Cardiology*. 2001; 37(5):1284–8. Epub 2001/04/13.
24. Hong MK, Mintz GS, Lee CW, Kim YH, Lee SW, Song JM, et al. Comparison of coronary plaque rupture between stable angina and acute myocardial infarction: a three-vessel intravascular ultrasound study in 235 patients. *Circulation*. 2004; 110(8):928–33. Epub 2004/08/18.

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