Original Article

Triggering Of Acute Coronary Syndromes By Physical Exertion And Anger

Masood Ali Akbar, Kashif Zafar, Abdul Rehman Abid, Rehan Riaz, Muhammad Azhar

ABSTRACT

Objectives: To assess the role of vigorous physical exertion and anger as triggers of acute coronary syndromes (ACS). **Materials And Methods:** This prospective observational study was conducted at the Punjab Institute of Cardiology, Lahore from April to September 2010. Two hundred patients admitted through emergency and out patient department were studied. Patients were questioned in detail about the circumstances surrounding the onset of acute symptoms. Anger was assessed according to the anger scale comprising of 7 points and physical activity was assessed according to activity scale also comprising of 7 points. **Results:** The mean age of the study population was 54.2±10.8 years. There were 149(74.5%) males and 51(25.5%) females. Diabetes mellitus occurred in 69(34.5%), hypertension 86(43%), smoking 71(35.5%) and dyslipidemia in 51(25.5%) patients. Majority of patients had low education status with primary education in 75(37.5%) and illiteracy in 74(37%) patients. Premonitory symptoms occurred in 92(46%) patients. Most patients 65(32.5%) presented to the hospital in 6-12 hours duration of onset of symptoms followed by 54(27%) patients presenting in 0-6 hours. Typical chest pain occurred in 166(83%) patients. Mostly patients 123(61.5%) had ST segment elevation myocardial infarction, followed by Non ST segment elevation myocardial infarction in 45(22.5%) and unstable angina in 31(15.5%) patients. The onset anger scale identified 25(12.5%) patients having associated anger at the time of onset of symptoms. According to anger scale, level 1 anger was observed in 5(2.5%), level 3 in 3(1.5%), level 4 in 4(2%), level 5 in 5(2.5%), level 6 in 6(3%) and level 7 in 2(1%) patients. The history of exertion at the time of onset of symptoms revealed that 95(47.5%) patients had level 1 exertion followed by level 2 exertion in 61(30.5%) and level 4 exertion in 14(7%) patients. **Conclusion:** This study confirms previous results and shows a graded exposure–response relationship between physical exertion intensity and triggering of AMI onset. The specific clinical and sociodemographic factors associated with physical exertion and anger suggest that different pathophysiological processes may be involved. **Key Words:** Acute myocardial infarction: triggering factors; anger; exertion.

INTRODUCTION

Cardiovascular diseases are presently the leading causes of death in industrialized countries and expected to become so in emerging countries by 2020.[1] Among these, coronary artery disease (CAD) is the most prevalent manifestation and is associated with high mortality and morbidity.[2] Physical exertion and episodes of anger occurring within 1–2 h of onset of symptoms have been identified as triggers of acute myocardial infarction (MI).[3-6] Physical and emotional stressors cause similar acute physiological changes to those seen in the morning period. These physiological changes can transiently increase the risk of plaque rupture and thrombosis and decrease the threshold for ventricular fibrillation.[7] The activities most commonly reported were emotional upset (18%), moderate physical activity (14%), heavy physical activity (9%), lack of sleep (8%), and overeating (7%).[7] In the ONSET study, heavy physical exertion (≥6 metabolic equivalents [METS]) in the hour before onset of MI symptoms was reported by 4.4% of patients and was associated with a relative risk of 5.9.3 In this population, heavy exertion can be considered to be the final component cause in 3.8% of cases, ie, ≈80% of the cases that

occurred within 1 hour of exertion were triggered by it.[3,8] In ONSET, 2.4% of patients reported anger that scored ≥5 on a 7-point anger scale in the 2-hour period before MI. This level of anger, which corresponded with "very angry, body tense, clenching fists or teeth" up to "furious or enraged," was associated with a transient 2-hour risk increase of 2.3 above baseline when the control was usual annual frequency.[4] This study was designed to evaluate the behavioural and emotional stimuli previously identified as triggers of acute MI and to assess the clinical and sociodemographic characteristics of patients who report possible triggers during the hours preceding symptom onset.

MATERIALS AND METHODS

This prospective observational study was conducted at the Punjab institute of cardiology Lahore from April to September 2010. Participants were two hundred patients admitted through emergency and out patient department. Information was obtained from medical notes about cardiovascular history and clinical factors during admission. The study was explained and informed consent was obtained, structured interview about triggering was taken. Patients were questioned in detail about the circumstances surrounding the onset of acute symptoms. In the light of previous literature we specifically focused on vigorous physical exertion with in one hour of symptom onset and anger with in two hours of symptoms on set. Vigorous physical exertion was defined as activity of at least six metabolic equivalents as used in earlier studies. The usual frequency of heavy exertion per week was inquired .Information regarding presence or absence premonitory symptoms, education, smoking; habitual physical activity was collected by questionnaire. Anger was assessed according to the anger scale as: (1) I am quick tempered, (2) I have a fiery temper, (3) I am a hot headed person, (4) I get angry when I am slowed down by others' mistakes, (5) I feel annoyed when I am not given recognition for doing good work, (6) I fly off the handle and (7) When I get angry, I say nasty things. Physical activity was assessed according to activity scale as: 1) Sleeping, 2) Lying, reclining Sunbathing, lying on a couch watching television, 3 and 4) Sitting, very light exertion, light exertion (METs 2–4), 5) Moderate exertion {(deep breathing, MET 5)}, 6) Vigorous exertion {(with panting, overheating, MET 6)}, 7) Heavy exertion (with gasping, much sweating, MET 7) Patients were included with diagnosis of ACS based on the presence of chest pain plus verification by diagnostic ECG changes (namely, new ST elevation >0.2mV in two contiguous leads in chest leads and >0.1 mV in two contiguous limb leads, ST depression >0.1mV in two contiguous leads in the absence of QRS confounders and new left bundle branch block or dynamic T wave inversions in more than one lead or Trop I measurement >0.1 or CK >twice the upper normal limit. Also patients with ability to complete a structured interview were included. Comorbid conditions that can influence either symptoms presentation or mood such as psychiatric illness, unexplained anemia, on going infection, inflammatory conditions, neoplasia and renal failure were excluded.

RESULTS

The mean age of the study population was 54.2±10.8 years. There were 149(74.5%) males and 51(25.5%) females. Diabetes mellitus occurred in 69(34.5%), hypertension 86(43%), smoking 71(35.5%) and dyslipidemia in 51(25.5%) patients. Table1. Majority of patients had low education status with primary education in 75(37.5%) and illiteracy in 74(37%) patients. Premonitory symptoms occurred in 92(46%) patients. Most patients 65(32.5%) presented to the hospital in 6-12 hours duration of onset of symptoms followed by 54(27%) patients presenting in 0-6 hours. Typical chest pain occurred in 166(83%) patients. Mostly patients 123(61.5%) had ST segment elevation myocardial infarction, followed by Non ST segment elevation myocardial infarction in 45(22.5%) and unstable angina in 31(15.5%) patients. The onset anger scale identified 25(12.5%) patients having associated anger at the time of onset of symptoms. According to anger scale, level 1 anger was observed in 5(2.5%), level 3 in 3(1.5%), level 4 in 4(2%), level 5 in 5(2.5%), level 6 in 6(3%) and level 7 in 2(1%) patients. Figure 1. The history of exertion at the time of onset of symptoms revealed that 95(47.5%) patients had level 1 exertion

followed by level 2 exertion in 61(30.5%) and level 4 exertion in 14(7%) patients. Figure1.

Table-1
Baseline characteristics of the study population

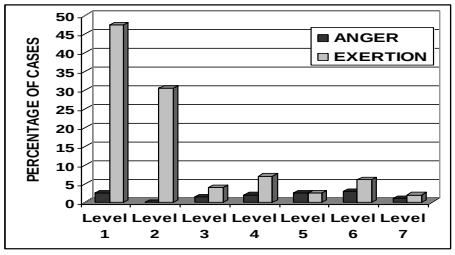
Characteristics	Numbers(Percentages)
Age mean years	54.2±10.8
Male	149(74.5%)
Female	51(25.5%)
Diabetes Mellitus	69(34.5%)
Hypertension	86(43%)
Smoking	71(35.5%)
Dyslipidemia	51(25.5%)
Obesity	49(24.5%)
Family history of IHD	72(36%)
Prior MI	11(5.5%)
Prior angina	51(25.5%)
Education Status	
None	74(37%)
Primary	75(37.5%)
Secondary	29(14.5%)
Graduation	22(11%)

Table -2
Presentation characteristics

CHARACTERISTICS	NUMBERS (PERCENTAGES)
Premonitory symptoms	92(46%)
Time of symptom onset	
0-6 hours	54(27%)
6-12 hours	65(32.5%)
12-18 hours	50(25%)
18-24 hours	27(13.5%)
Typical chest pain	166(83%)
STEMI	123(61.5%)
NSTEMI	45(22.5%)
Unstable angina	31(15.5%)

STEMI= ST segment elevation myocardial infarction. NSTEMI= Non-ST segment elevation myocardial infarction.

Figure-1
Anger and exertion levels at the time of onset of symptoms



DISCUSSION

The purpose of this investigation was to discover whether the behavioural and environmental stimuli previously identified as triggers of acute MI are relevant to the broad spectrum of ACS and to assess the clinical and sociodemographic characteristics of patients who report possible triggers during the hours preceding symptom onset. The proportion of patient who reported vigorous physical exertion in one hour preceding symptom on set was 6%. Previous studies of acute MI have reported heavy exertion in 4.4%,6.4% and 7.5%.[5,6,10] The prevalence of anger over 2hours before symptom onset(12.5%)was greater than reported in the ONSET and Stockholm Heart Epidemiology Program (SHEEP) studies both of which had prevalence less than 2.5%.[4,6] This is because of our use of broader inclusion criteria for anger episodes. Previous studies have analyzed only high levels of anger, when corresponding criteria were applied to present data set, prevalence of anger fell to 3.5%,but the number of cases was too small for further analysis.[6] The patients with ACS in this study sample were relatively young. With only moderate risk profile as measured with Global Registry of Acute Coronary Events (GRACE) index.[11] There was also higher proportion of STEMI than of NSTEMI/UA than that reported in recent studies[12] probably a result of requirement that patients could not recall a specific time of onset. Both types of triggers were more commonly associated with STEMI than with other forms of ACS. Premonitory symptoms occurred in 46% patients it is possible that a premonitory symptom signals an important biologic event such as plaque rupture or the beginning of thrombosis.[13] The mechanisms underlying these patterns of triggering are not fully understood. Physical exertion leads to an acute increase in sympathetic activity, release of catecholamines and haemodynamic responses that may lead to plaque rupture. Catecholamines stimulate surface expression of adhesion molecules, and concentration of interleukin 6 in the circulation rises notably.[14] Acute mental stress also stimulates inflammatory cytokines, lymphocyte adhesion molecule expression and platelet activation endothelin-1.[15,16] Platelet reactivity to mental stress has been positively correlated with hostile personality traits[17] and this may contribute to

association with anger. Although anger triggering was related to presentation with STEMI, however, it was not associated with the development of Q-wave infarction, heart failure or magnitude of troponin T release. This suggests that anger may have transiently influenced physiology and implicates a possible role of vascular spasm. Boltwood et al[18] reported that anger recall during catheterization induced coronary vasoconstriction in stenosed vessels, and this phenomenon may translate in to clinical risk of ACS. Emotional stress has been shown in ambulatory monitoring studies to induce transient myocardial ischaemia.[13] Another relevant mechanism is disturbance of autonomic function leading to arrhythmia.[19] Anger in the hours immediately preceding symptom onset was associated with younger age and greater socioeconomic deprivation the age effect suggests that people who are prone to anger are less likely to survive into old age a relation ship between anger triggering and lower socioeconomic status defined by education was described by Onset study.[20] This result is consistent with notion that emotional factors are responsible in part for the socioeconomic gradient in risk of coronary heart disease. This study has some limitations. The sample size was relatively small and prevented us from dividing out Non ST Elevation myocardial infarction from unstable angina. Exclusion of patients with co-morbidities that can affect symptoms presentation, mood or cardiac enzymes led to restriction of sample to people with relatively low risk. Patients' retrospective reports of their experiences before symptom onset may be biased or influenced by the salience of the situation leading to overestimation during hazard period.[21] The strengths of the investigation include assessment of different types of ACS, characterization of patients in terms of multivariate clinical risk, and measurement of troponin in the majority of patients. The results indicate that triggering by exertion and anger is relevant not only to acute MI but also to other presentations across the range of ACS. The distinctive clinical and sociodemographic profiles of triggering by exertion and anger suggest that different patients are at risk from these factors .It remains to be seen whether the pathophysiological processes underlying triggering by physical exertion and anger also vary.

CONCLUSION

This study confirms previous results and shows a graded exposure–response relationship between physical exertion intensity and triggering of AMI onset. The specific clinical and sociodemographic factors associated with physical exertion and anger suggest that different pathophysiological processes may be involved.

REFERENCES

1. Murray CJ, Lopez AD. Alternative projections of mortality and disability by cause 1990–2020: Global Burden of Disease Study. Lancet 1997; 349: 1498–1504.
2. Bassand J, Hamm CW, Ardissino D, Boersma E, Budaj A, Ferna´ndez-Avile´s F, et al. Guidelines for the diagnosis and treatment of non-ST-segment elevation acute coronary syndromes. Eur Heart J 2007;28:1598–1660
3. Mittleman MA, Maclure M, Tofler GH, et al. Triggering of acute myocardial infarction by heavy physical exertion: protection against triggering by regular exertion. Determinants of Myocardial Infarction Onset Study Investigators. N Engl J Med 1993;329:1677–83
4. Mittleman MA, Maclure M, Sherwood JB, et al. Triggering of acute myocardial infarction onset by episodes of anger. Circulation 1995; 92:1720-5.
5. Willich SN, Lewis M, Lowel H, et al. Physical exertion as a trigger of acute myocardial infarction. Triggers and Mechanisms of Myocardial Infarction Study Group. N Engl J Med 1993;329:1684–90.
6. Mo¨ller J, Hallqvist J, Diderichsen F, et al. Do episodes of anger trigger myocardial infarction? A case-crossover analysis in the Stockholm Heart Epidemiology Program (SHEEP). Psychosom Med 1999;61:842–9.
7. Muller JE, Abela GS, Nesto RW, Tofler GH. Triggers, acute risk factors and vulnerable plaques: the lexicon of a new frontier. J Am Coll Cardiol. 1994; 23:809–813.
8. Rothman KJ, Greenland S. Modern Epidemiology. 2nd ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 1998.
9. Hallqvist J, Moller J, Ahlbom A, et al. Does heavy physical exertion trigger myocardial infarction? A case crossover analysis nested in a

population based case referent study. Am J Epidemiol 2000;151:459–467.
10. Rosengren A, Wallentin L, Gitt A K, et al. Sex, age, and clinical presentation of acute coronary syndromes. Eur Heart J 2004; 25: 663–670.
11. Eagle KA, Lim MJ, Dabbous OH, et al. A validated prediction model for all forms of acute coronary syndrome: estimating the risk of 6 month postdischarge death in an international registry. JAMA 2004; 291: 2727–2733.
12. Fox KA, Goodman SG, Klein W, et al. Management of acute coronary syndromes: variations in practice and outcome: findings from the Global Registry of Acute Coronary Events (GRACE). Eur Heart J 2002; 231:1177–1189.
13. Gullette EC, Blumenthal JA, Babyak M, et al. Effects of mental stress on myocardial ischemia during daily life. JAMA 1997; 277:1521–1526.
14. Shephard RJ. Adhesion molecules, catecholamines and leucocyte redistribution during and following exercise. Sports Med 2003;33:261–284.
15. Steptoe A, Willemsen G, Owen N, et al. Acute mental stress elicits delayed increases in circulating inflammatory cytokine levels. Clin Sci (Colch) 2001; 101:185–192.
16. Von Kanel R, Mills PJ, Fainman C, et al. Effects of psychological stress and psychiatric disorders on blood coagulation and fibrinolysis: a biobehavioral pathway to coronary artery disease? Psychosom Med 2001;63:531–544.
17. Markovitz JH, Matthews KA, Kriss J, et al. Effects of hostility on platelet reactivity to psychological stress in coronary heart disease patients and in healthy controls. Psychosom Med 1996; 58:143–149.
18. Boltwood MD, Taylor C B, Burke M B, et al. Anger report predicts coronary artery vasomotor response to mental stress in atherosclerotic segments. Am J Cardiol 1993; 72: 1361–1365.
19. Hemingway H, Malik M, Marmot M. Social and psychosocial influences on sudden cardiac death, ventricular arrhythmia and cardiac autonomic function. Eur Heart J 2001; 22: 1082–1101.
20. Mittleman M A, Maclure M, Nachnani M, et al. Educational attainment, anger, and the risk of triggering myocardial infarction onset. Arch Intern Med 1997; 15: 7769–775.
21. Strike PC, Steptoe A. Behavioral and emotional triggers of acute coronary syndromes: a systematic review and critique. Psychosom Med 2005; 67179–186.

AUTHORS

- **Masood Ali Akbar**
  FCPS(Med), FCPS (Card)
  Senior Registrar Cardiology,
  Punjab Institute of Cardiology, Lahore
  Kashif Zafar
  FCPS (Med), FCPS (Card)
  Senior Registrar Radiology
  Punjab Institute of Cardiology, Lahore
- **Abdul Rehman Abid**
  FCPS (Cardiology)
  Senior Registrar Cardiology
  Punjab Institute of Cardiology, Lahore
- **Rehan Riaz**
  M.B. B.S
  Medical Officer
  Punjab Institute of Cardiology, Lahore
- **Muhammad Azhar**
  MRCP (UK), FACC
  Professor& Chief of Cardiology
  Punjab Institute of Cardiology, Lahore

Correspondence Address
Kashif Zafar, FCPS (Med), FCPS (Card)
Senior Registrar Cardiology,
Punjab Institute of Cardiology, Lahore
E-mail: drkashifzafar@hotmail.com