Bull Yamaguchi Med Sch 31 : 79-85, 1984

Physical Activities Associated with Angina Pectoris Before Myocardial Infarction and the Onset of Myocardial Infarction

Masako Matsuda

Second Department of Internal Medicine, Yamaguchi University School of Medicine, Ube, Yamaguchi 755, Japan

(Received May 1, 1984)

Abstracts One hundred and ninety-seven patients with a history of myocardial infarction were interviewed to evaluate the incidence of angina pectoris and the physical activity precipitating angina before myocardial infarction, and the mode of physical activity at the onset of myocardial infarction. Ninety-ewo patients had no angina before infarction, whereas 105 did. In 105 patients, 58 had a chronic stable angina without a change of pattern of angina before infarction, while 22 noticed worsening of their symptoms within 2 weeks before infarction. Twenty five patients had new-onset angina within 2 weeks before infarction. In 92 patients without angina before infarction, myocardial infarction occurred during hard exertion in 10, mild exertion in 43, at rest in 28 and during sleep in 11. In 58 patients with chronic stable angina, 47 had angina on exertion, 7 at rest and 4 on both. However, the subsequent infarction occurred during hard exertion in 9, mild exertion in 16, at rest in 25, and during sleep in 8. In these patients without the worsening of symptoms or new onset of angina within 2 weeks before infarction, myocardial infarction occurred unpredictably or differently from the mode of physical activity that provoked angina.

Key Words: Angina pectoris, Myocardial infarction

Introduction

Angina pectoris is a symptom of myocardial ischemia resulted from an imbalance between oxygen supply and demand, generally seen in patients with coronary arterial obstructive disease[1]. Exercise is the commonest precipitating factor and angina during exercise has been considered to be related to inability of a fixed coronary reserve to meet the increased myocardial oxygen demands[2]. The pathogenesis of myocardial infarction is controversial. It has been reported that the physical activity does not play a role in the genesis of myocardial infarction[3].

In this study, we analyzed the physical activity precipitating angina pectoris before myocardial infarction and the physical activity at the onset of myocardial infarction, in patients with symptomatically documented myocardial infarction. And we observed the correlation of physical activity between angina before infarction and the subsequent myocardial infarction.

Methods

This study comprises all patients admitted to the hospital with the documented diagnosis of myocardial infarction. We excluded the patients with associated congenital heart disease, valvular heart disease, reattack of infarction or if a specific date of infarction could not be determined. Myocardial infarction was defined by the presence of compatible clinical history, and a diagnostic ECG pattern of transmural necrosis or a characteristic increase in appropriate serum enzyme determination. In all cases, clinical history was obtained from the patients by a physician (M.M.) involved in this study.

The coding of symptoms before myocardial infarction was designed as follows: no angina before myocardial infarction (Group Ⅰ), chronic stable angina (Group Ⅱa), chronic angina with worsening of symptoms within the 2 weeks before infarction (Group Ⅱb), and new onset angina within the 2 weeks before infarction (Group Ⅲ). Chest pain that occurred for the first time or altered its character within 2 weeks prior to infarction was defined as an unstable angina, a procedure of acute infarction.[4-6]

Types of physical activity at the onset of myocardial infarction was divided into 4 groups; sleeping, rest, mild activity and hard activity[3]. Rest was defined as lying or sitting without any active movement. Mild activity was defined as ordinary mild activity such as light house working, walking about, standing, sitting in the office, getting out of bed and attending a meeting. Hard activity was defined as moderate to hard activity such as running upstairs, playing a base ball or unusual field work.

Types of physical activity precipitating angina before infarction were devided into 2 groups; rest and effort. The group of effort angina consisted of the patients with angina provoked by the activity which increased the myocardial oxygen onsumption, while the group of resting angina dfd oi the patients wh odevelopedangina at rest. Statistical analysis was performed with the Student'st test and the χ^2 test.

Results

One hundred and ninty-seven patients, 173 men and 24 women, mean age of 54±8 years, were divided into 4 groups according to the presence or absence of angina and the type of angina before myocardial infarction (Fig. 1). In the series of 197 patients, 92 patients had no angina before myocardial infarction (Group Ⅰ) and the remaining 105 patients had angina before infarction. Of these 105 patients, 80 patients (Group Ⅱ) had angina more than 2 weeks before infarc-

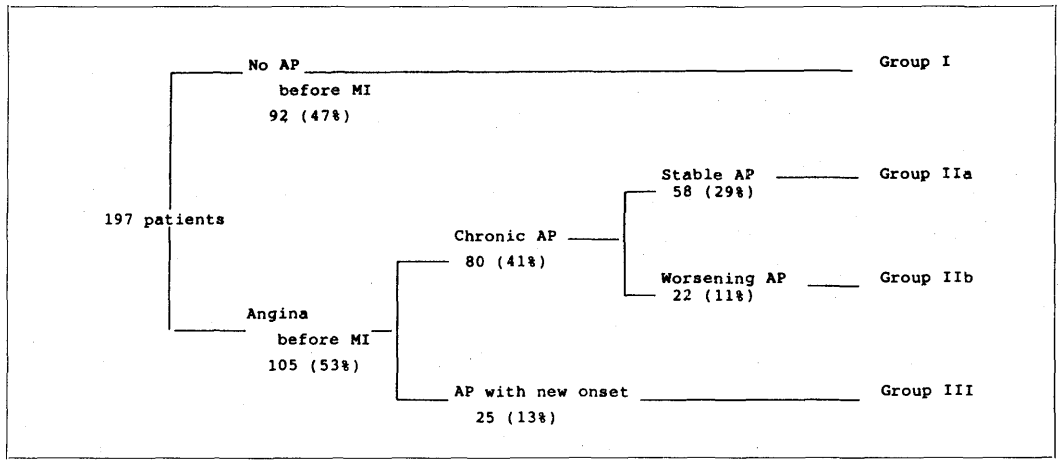

Fig. 1 Diagram indicating the breakdown of the 197 patients into the Groups Ⅰ, Ⅱa, Ⅱb, Ⅲ. AP= angina pectoris; MI=myocardial infarction.

tion and of those, 58 (Group Ⅱa) had no change in the pattern of angina before infarction, while 22 (Group Ⅱb) described worsening angina within the 2 weeks prior to infarction. Twenty five patients developed new onset of angina within 2 weeks prior to infarction (Group Ⅲ). The total number of patients with unstable angina (Group Ⅱb and Group Ⅲ) was 47.

Physical activities at the onset of myocardial infarction in Groups Ⅰ, Ⅱa, Ⅱb and Ⅲ are shown in Table 1. The relationship between each group and the physical activity at the onset of myocardial infarction was independent by chi-square distribution. Of 92 patients of Group Ⅰ, myocardial infarction occurred during hard exertion in 10, mild exertion in 43, at rest in 28 and during sleep in 11

Table 1 Physical activities at the onset of myocardial infarction in each group

Groups	Activities at the onset of infarction				Total
	Sleep	Rest	Mild	Hard	
Ⅰ	11	28	43	10	92
Ⅱa	8	25	16	9	58
Ⅱb	8	6	5	3	22
Ⅲ	6	10	7	2	25
Total	33	69	71	24	197

Table 2 Types of angina pectoris and relationships to the mode of the physical activities at the onset of infarction in chronic stable angina group (Group Ⅱa)

Types of angina	Activities at the onset of infarction				Total
	Sleep	Rest	Mild	Hard	
Effort	4	22	13	8	47
Rest	2	2	2	1	7
Both	2	1	1	0	4

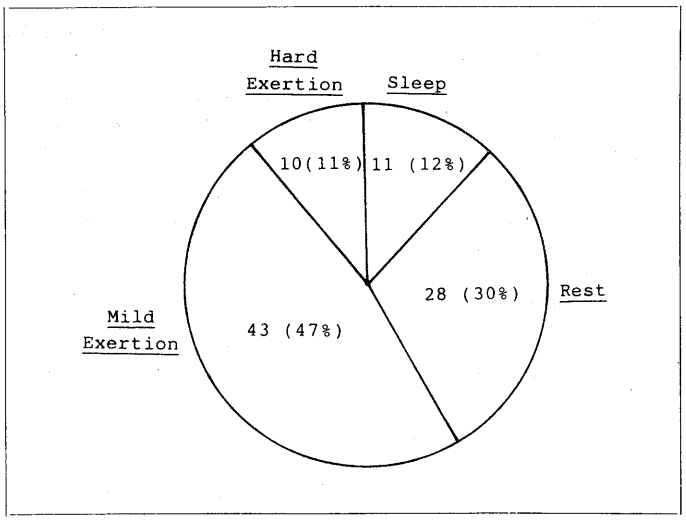

Fig. 2 Physical activities at the onset of myocardial infarction in patients without angina before infarction (Group I).

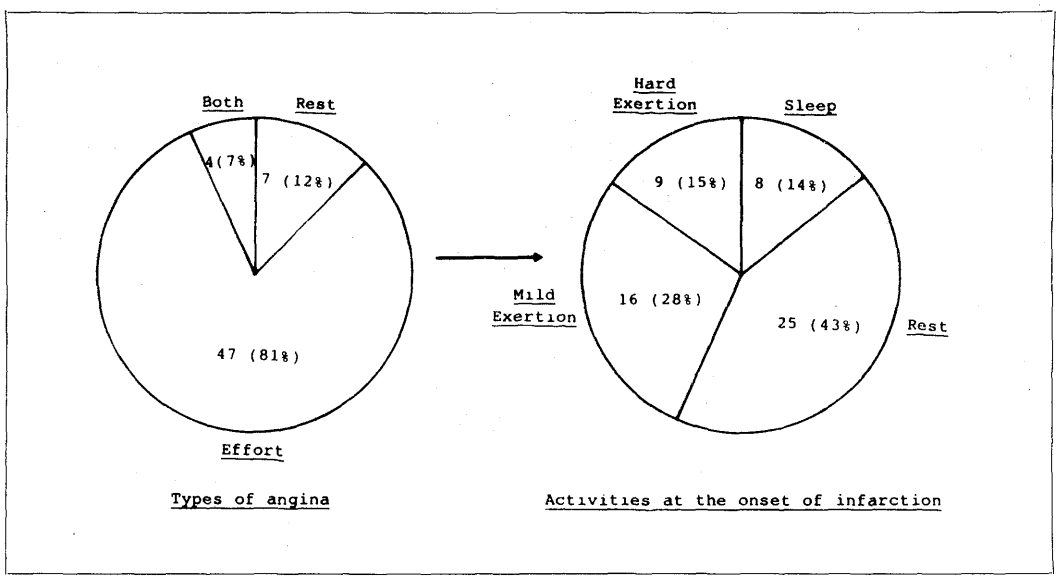

Fig. 3 Types of angina and relationships to the mode of physical activities at the onset of infarction in chronic stable angina group (Group Ⅱ a).

(Fig. 2). Of 58 patients of Group Ⅱ a, 47 had angina on exertion, 7 at rest and 4 on both. However, the subsequent infarction occurred during hard exertion in 9, mild exertion in 16, at rest in 25 and during sleep in 8 (Table 2, Fig. 3). Of patients with unstable angina (Group Ⅱ b and Group Ⅲ), hard exertion provoked myocardial infarction in 5, mild exertion in 12, rest in 16 and sleep in 14.

The duration of symptoms prior to infarction of 105 patients with angina before myocardial infarction is shown in Fig. 4. Twenty-nine patients developed angina more than 1 year before the infarction. Fourteen patients developed the symptom of angina between 3 months to 1 year before infarction .Sixty-two patients, 59% of the total patients with angina before infarction, developed angina 3 months prior to infarction. In 18 of 22 patients who had chronic angina with worsening within 2 weeks before myocardial infarction, the instability occurred within 1 week of infarction.

The time of onset of myocardial infarction was known in 189 of 197 patients (Table Ⅲ). There was a preponderence of attacks in the evening (6PM-0AM) and less attacks in the night (0AM-6AM). Fifty per cent of attacks occurred during the daytime (6AM-6PM), and the rest began during the night (6PM-6AM).

The age at the time of myocardial infarction is shown in Table IV. The mean age was 54 ± 8 years in male, 59 ± 9 in female, respectively. Female patients developed myocardial infarction in older age than men ($p < 0.01$). The peak of the frequency of the attack was in fifties in men and in sixties in women.

Discussion

The incidence of angina pectoris before myocardial infarction is reported previously [6-10]. Yater et al[7]. observed 50.6 per cent of patients with myocardial infarction had no

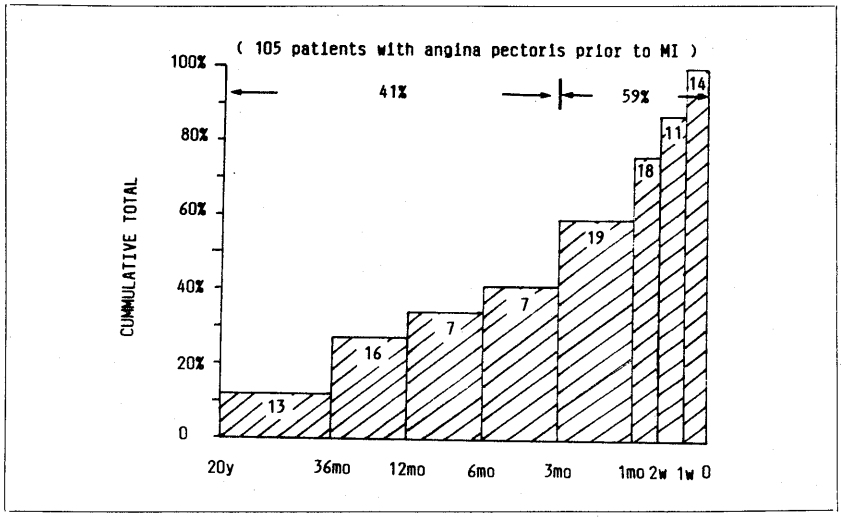

Fig. 4 Duration of angina pectoris prior to onset of myocardial infarction. MI=myocardial infarction; mo=months; w=weeks; y=years.

Table 3 Time of onset of myocardial infarction

	Attacks	Incidence
6am–Noon	53	28.0%
Noon–6pm	41	21.7%
During the daytime (6am–6pm)	94	49.7%
6pm–0am	63	33.3%
0am–6am	32	16.9%
During the night (6pm–6am)	95	50.2%

Table 4 Age at the occurrence of MI

	≦40	41–50	51–60	61–70	71≦
Male	12	42	83	35	1
Female	1	3	6	13	1

previous history of angina. Solomon and his associates[8] reported the incidence of angina, before myocardial infarction was 41 per cent 81 per cent of which had new symptoms or worsening of symptoms that preceded myocardial infarction by a period not exceeding 2 months. Midwall and his group[4] showed that the preexisting angina more than 2 weeks before myocardial infarction was observed in 35 per cent of patients with infarction. Harper et al.[9] found that 52 per cent of patients had angina before myocardial infarction, and 31 per cent of patients had chronic angina, which was defined as angina present for more than one month prior to myocardial infarction. In the present study, 92 patients had no angina before myocardial infarction, whereas 105 did. Of 105 patients, 58 had a chronic stable angina ,while 22 noticed worsening of their symptoms within2 weeks before infarction. Twenty-five patients had new onset angina within 2 weeks before infarction.

Many factors may account for the variable range of incidence of angina pectoris before myocardial infarction, including methods of the study, variability in diagnostic criteria, and sampling from different populations. The presence or absence of angina before myo-

cardial infarction, and the mode of physical activity precipitating angina and at the onset of myocardial infarction are subjective assessments both on the part of patient and the interviewer. In this study, the interview with each patient was performed by one physician (M.M.). The incidence of angina in this study are consistent with the tendency of the previous studies[5-7]. The mode of physical activity at the onset of myocardial infarction was as follows; 24 on hard exertion, 71 are mild exertion, 69 at rest, and 33 during sleeping. Phipps[11] reported that the onset of myocardial infarction was not related to physical stress in 60 per cent of their cases. Master et al[3,12]. also observed that 53 per cent of myocardial infarction occurred either at rest or while sleeping. In their study on the time of onset of 722 attacks, 52 per cent of the attacks began during the daytime (7 A.M. to 7 P.M.), and 48 per cent during the night (7 P.M. to 7 A.M.). The present findings on the mode of physical activity at the onset of myocardial infarction were compatible with the previous results.

In 92 patients without angina before infarction, myocardial infarction occurred during hard exertion in 10, mild exertion in 43, at rest in 28 and during sleep in 11. In 58 patients with chronic stable angina, 47 had angina on exertion, 7 at rest and 4 on both. However, the subsequent infarction occurred during hard exertion in 9, mild exertion in 16, at rest in 25, and during sleep in 8. In patients with chronic stable angina, most of the attacks of angina were precipitated by the increased physical activity and subsequent myocardial infarction occurred on various degree of physical activity. Unstable angina has been regarded as a premonitory sign of impending acute myocardial infarction, and defined as either new onset angina or an increased in severity of symptoms in patients with chronic angina prior to infarction. Patients with unstable angina can be predicted and managed to alleviate the development of myocardial infarction[5,13-15]. However, 150 out of 197 patients had either no angina or chronic stable angina before myocardial infarction. In these patients, myocardial infarction occurred unpredictably in spite of the presence or absence of angina before infarction. The degree of physical activity precipitating angina before myocardial infarction was different from that at the onset of myocardial infarction.

Shub et al[16]. made the observation that progression to occlusion occurs in relation to the degree of stenosis at first study and that this can be observed independently from time. Kramer et al[17]. suggested that two processes, one time-dependent and one time-independent, determine the rate of progression in coronary atherosclerosis. Nonocclusive progression occurred in relation to time, while progression to occlusion did not depend directly on time. Postmortem studies also reveal a duality of the progressive pathologic process leading to fatal acute myocardial infarction[18-21]. Plaque formation develops in a time-dependent, chronic fashion until rupture of the plaque leads to vessel occlusion with atheromatous material or thrombus in an acute, time-independent manner. In vivo, the successful thrombolytic reperfusion of an acutely thrombosed coronary artery often reveals a significant, fixed, underlying obstruction[22], again suggesting that two pathologic processes are involved. The present observation of clinical symptoms may also support that acute occlusive event of myocardial infarction could be caused by the different mechanism associated or unassociated with the progression of athe rosclerosis, rather than the termination of coronary atherosclerotic progression.

I thank Dr. Reizo Kusukawa, Professor of the Second Department of Internal Medicine, Yamaguchi University School of Medicine, for his warm guidance and reviewing through this paper, and

Dr. Yasuo Matsuda for his helpful advice and great cooperation. I also deeply appreciate all the members of the Second Department of Internal Medicine, Yamaguchi University School of Medicine for their understanding on my work.

References

1) Gorlin, R.: Pathophysiology of cardiac pain. *Circulation*, 32 : 138-148, 1965.
2) Friedberg, C.K.: Angina pectoris. *Circulation*. 46 : 1037-1047, 1972.
3) Master, A.M.: Dack, S., Jaffe, H.L.: Activities associated with the onset of acute coronary artery occlusion. *Am. Heart J.*, 18 : 434-443, 1939.
4) Alonzo, A.A., Simon, A.B., Feinleib, M.: Prodromata of myocardial infarction and sudden death. *Circulation*, 52 : 1056-1062, 1975.
5) National Cooperative Study Group: Unstable angina pectoris: National Cooperative Study Group to compare medical and surgical therapy. Ⅰ. Report of protocol and patient population. *Am. J. Cardiol.*, 37 : 896-901, 1976.
6) Midwall, J., Ambrose, J., Pichard, A., Abedin Z., Herman, M.V.: Angina pectoris before and after myocardial infarction. *Chest*, 81 : 681-686, 1982.
7) Yater, W.M., Traum, A.H., Spring, S., Brown, W.G., Fitzgerald, R.P., Murray, A.G., Wilcox,, B.B.: Coronary artery disease in men eighteen to thirty-nine years of age. *Am. Heart J.*, 36 : 334-372, 1948.
8) Solomon, H.A., Edwards, A.L., Killip, T.: Prodromata in acute myocardial infarction. *Circulation*, 40 : 463-471, 1969.
9) Harper, R.W., Kennedy, G., DeSanctis, R.W., Hutter, A.M.: The incidence and pattern of angina prior to acute myocardial infarction: A study of 577. *Am. Heart J.*, 97 : 178-183, 1979
10) Master, A.M., Dack, S., Jaffe, H.L.: Premonitory symptoms of acute coronary occlusion; a study of 260 cases. *Ann. Intern. Med.*, 14 : 1155-1165, 1941.
11) Phipps, C.: Contributory causes of coronary thrombosis. *J.A.M.A.*, 106 : 761-762, 1936.
12) Master, A.M., Dack, S., Jaffe, H.L.: Factors and events associated with onset of coronary artery thrombosis. *J.A.M.A.*, 109 : 546-549, 1937.
13) National Cooperative Study Group: Unstable angina pectoris: National Cooperative Study Group to compare surgical and medical therapy. Ⅱ. In-hospital experience and initial follow-up results in patients with one, two and three vessel disease. *Am. J. Cardiol.*, 42 : 839-848, 1978.
14) Cairns, J.A., Fantus, I.G., Klassen, G.A.: Unstable angina pectoris. *Am. Heart J.*, 92 : 373-386, 1976.
15) Victor, M.F., Likoff, M.J., Mintz, G.S., Likoff, W*: Unstable angina pectoris of new onset: a prospective clinical and arteriographic study of 75 patients. *Am. J. Cardiol.*, 47 : 228-232, 1981.
16) Shub, C., Vlietstra, R.E., Smith, H.C., Fulton, R.E., Elveback, L.R.: The unpredictable progression of symptomatic coronary artery disease .A serial clinical angiographic analysis. *Mayo Clin. Proc.*, 56 : 155-160, 1981.
17) Kramer, J.R., Kitazume, H., Proudfit, W.L., Matsuda, Y., Goormastic, M., Williams, G.W., Sones, F.M.: Segmental analysis of the rate of progression in patients with progressive coronary atherosclerosis. *Am. Heart J.*, 106 : 1427-1431, 1983.
18) Horie, T., Sekiguchi, M., Hirosawa, K.: Coronary thrombosis in pathogenesis of acute myocardial infrction. Histopathological study of coronary arteries in 108 necropsied cases using serial selection. *Br. Heart J.*, 40 : 153-161, 1978.
19) Chapman, I.: Morphogenesis of occluding coronary artery thrombosis. *Arch. Pathol.*, 80 : 256-261, 1965.
20) Bouch, D.C., Montgomery, G.L.: Cardiac lesions in fatal cases of recent myocardial ischemia from a coronary care unit. *Br. Heart J.*, 32 : 795-803, 1970.
21) Ridolfi, R.L., Hutchins, G.M.: The relationship between coronary artery lesions and myocardia. infarcts: Ulceration of atherosclerotic plaques precipitating coronary thrombosis. *Am. Heart J.*, 93 : 468-486, 1977.
22) Rutsch, W., Schartl, M., Mathey, D., Kuck, K., Merx, W., Dorr, R., Rentrop, P., Blanke, H.: Percutaneous transluminal coronary recanalization: Procedure, results, and acute complication. *Am. Heart J.*, 102 : 1178-1181, 1981.