PRODROMES IN ACUTE MYOCARDIAL INFARCTION*

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Acute myocardial infarction apparently strikes its victims with dramatic suddenness. Although the underlying pathologic process is almost invariably atherosclerosis of coronary arteries, the morbid sudden event or combination or chain of events precipitating myocardial infarction are not well defined. There has been considerable research in identifying the risk factors which may singly or in combination influence the genesis of coronary atherosclerosis. Such risk factors include heredity, hyperlipidemia, diabetes mellitus, hypertension, smoking, physical inactivity and obesity. The effect of these factors is believed to be additive. The presence of these factors, however, does not imply that significant coronary artery disease has already developed. These risk factors may be determinants of atherogenesis, but they certainly may not be the cause of final morbid event provocative of acute myocardial infaction. In certain cases, such as diabetes mellitus and hypertension, even effective control of the underlying risk factor may not significantly influence the evaluation, incidence and severity of coronary artery disease. Hyperlipidemia may need paediatric through geriatric care.

Prehospital mortality of acute myocardial infarction is high ranging from 25 to 60 per cent (Table 1) (Moss and Goldstein,1 972). Since the highest mortality figures embrace first 12 to 24 hours (Fulton et al., 1969), a 24 hour delay in hospitalisation would, therefore, exclude most of the early deaths. The hospital mortality figures are, therefore, a gross under-estimate of true mortality figures determined by time lapse between the onset of disease and admission of the patient to the hospital.

Table 1: Acute Myocardial Infarction

1.	Pre-hospital Mortality (mostly sudden deaths) 84% in first 12 to 24 hours.	25 to 60 per cent.
2.	Hospital Mortality (Before C.C.U.)	30 to 50 per cent
3.	Hospital Mortality (in C.C.U.)	15 to 30 per cent

Since the advent of coronary care units, the overall mortality of hospitalised patients is about 15 to 30 per cent, averaging 20 per cent (Moss and Goldstein, 1972). Most of the deaths in hospitalised patients occur during the first week, the largest percentage of these during the first 24 to 48 hours. There are practically no deaths after the first three weeks (Lukl, 1971).

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The coronary care units have reduced the hospital mortality rate by 30 to 50 per cent. Therefore a large segment of patients suffering acute myocardial infarction still die inspite of the sophisticated, emotionally charged, electronically stunning environment with constant monitoring of heart rate, rhythm and blood pressure. Sudden deaths still account for about 50 to 60 per cent of total deaths due to coronary eartery disease and is, therefore, the most serious and thereatening syndrome of coronary artery disease. Several recent studies have emphasised that mobile life support stations or mobile coronary care units have made little inroads on the mortality figures because of the patient delay, most sudden deaths occurring during the first hour. The majority of deaths in acute myocardial infarction are sudden deaths which are electrical deaths due to ventricular fibrillation. These deaths rates are difficult to subdue with active therapeutic plans. Their control needs preventive measures.

There appear to be several types of symptoms and signs which may have ominous significance. Such apparently vague clinical expressions afflict the patients days and weeks before they are stricken by acute myocardial infarction.

This presentation is a retrospective study of 100 patients who survived acute transmural myocardial infarction. All patients had typical and characteristic clinical profile, evolutionary electrocardiographic changes and elevation of SGOT and LDH.

Premonitory clinical expressions shown by these patients are given in Table 2.

Table 2: Premonitory Clinical Expressions

Clinical Expression	*Male*	*Female*
1. Undue Fatigue and weakness	72	12
2. Palpitation	23	2
3. Burning Sensation, generalised	7	1
4. Vague and atypical chest pain	43	7
5. Pain in the interscapular region.	9	—
6. Indigestion.	27	4
7. Excessive sweating.	16	3
8. Paraesthesias or weakness in the arms.	11	2
9. Exertional dynspnoea	69	10
10. Undue tendency to sleep at inappropriate occasions.	67	12
11. Giddiness	13	2
12. Syncope, fainting.	2	—
13. Hemiplegia	1	—
14. Sudden hypotension.	3	1
15. Change in behaviour.	53	9
16. Premature aging.	33	3
17. Gallop rhythm (ventricular)	9	—
18. E.C.G. Changes.	7	—

The *age and sex* distribution of these patients is given in Table 3 and 4. Common prodromes experienced by these patients are given in Table 5.

Table 3: Survivors of AMI

100 Pts: Age Distribution		
Age in years	*Males*	*Females*
31 to 40	4	—
41 to 50	30	5
51 to 60	37	6
61 to 70	14	4

Table 4: Survivors of AMI

100 Pts. Sex Distribution			
Ant. AMI		Inf. AMI	
Males	*Females*	*Males*	*Females*
60	7	25	8
Total Males:	85		
Total Females:	15		

Table 5 Common Prodromes

1. Fatigue and weakness.
2. Autonomic dysfunction
   —palpitation
   —sweating
3. Exertional dyspnoea
4. Indigestion; gas, heart-burn, vague epigastric discomfort.
5. Vague chest pain.
6. Paraesthesias in the arm or weakness of the arm/arms.
7. Undue tendency to sleep.
8. Change in behaviour.
9. Premature aging.
10. Gallop rhythm.
11. E.C.G. changes.

Risk factors present in these patients are given in Table 6.

Table 6: Predisposing Conditions

Disease	*Males*	*Females*
1. Hypertension	67	11
2. Diabetes mellitus	17	6
3. Raised serum cholesterol (⩾ 300 mg/100 ml)	74	9
4. Obesity	31	12
5. Excessive smoking. (Bidi or Cigartes, Hooka)	79	5
6. Ambitious, competitive and work addict.	62	2

Discussion

Fatigue and exertional dyspnea are probably due to impaired left ventricular performance causing decreased cardiac output. Coronary athero sclerosis in these patients has already caused borderline cardiac reserve unable to withstand any physical or emotional stress. Possibly, depletion of cardiac catecholamines occurs through years of stress occasioned by Type A personality, abnormal behavior patients and work addiction. These patients have been working beyond their capaccities for years without rest, recreation, leisure and holiday. Others, more affluent care-free landlords were overeating, smoking heavily and trying to obtain relief from boredom by indulging in various types of addicition. Myocardium, 'depleted of catecholamines and hence sympathetic support and drive,' works at a lower ventricular function curve with increased left ventricular end-diastolic pressure, increased wall tension, reduced complicance and increased myocardial oxygen consumption. These haemodynamic abnormalities may cause ventricular gallop which was heard in 9 out of 15 patients who were seen with suspect symptoms before the critical event.

Palpitation may be due to premature beats or paroxysmal tachyarrhythmias originating in the myocardium with compromised blood supply. Such regional myocardial ischaemia may facilitate electrical instability and automaticity.

Indigestion may be referred cardiac pain in the epigastrium or may be due to circulatory insufficiency due to reduced cardiac output or associated mesenteric atherosclerosis. It may, however, be due to excessive indulgence in smoking and overuse of tea, coffee or alchol.

Paraesthesias in the arms may be atypical angina equivalent or perhaps due to reduced cerebral circulation.

Undue tendency to sleep at odd hours and at odd places is probably due to cumulative fatigue due to years of overwork with no leisure and recreation. It may be due to reduced vertebrobasilar circulation due to reduced cardiac output causing impaired functioning of reticular activating system.

Giddiness, faintness and hemiparesis are neurological expressions of regional or generalised cerebral circulatory deficit. It should, therefore be a routine to take ECG in all patients who complain of such symptoms.

Angina pectoris in its typical or atypical form may occur lasting for a short time. It may take the form of toothache, pain in the jaw, pain in the interscapular region or pain localised to a small region in the arm. In one patient, the presentation was generalised burning sensation. Many patients sought relief by pressing the affected area and several patients presented with a tight binder around the affected arm cliamed to have subdued the arm pain. In some, gentle tapping in the interscapular region decreased the severity of chest and arm pain. These physical modalities of treatment so often witnessed in people coming from rural areas may be acting by competitive inhibition of pain subserving pathways.

The mild anginal pain may be dismissed as trivial, being due to indigestion, gas or muscle strain. Any acute pain of any intensity localised between ambilicus and the jaw needs an electrocardiogram. One should be cautions and respectful to an anginal pain of sudden onset, stable angina which has recently undergone deterioration such as increase in severity, intensity and/or duration and inadequate or lack of response to nitroglycerine. Such anginal pain is even more serious and disastrous due to increasing risk of electrical instability and progression towards severe infarction. Such pain implies myocardial infarction whether or not such infarct is revealed by electrocardiogram or enzyme profile.

If these premonitary clinical expressions are kept in mind, many patients with prospective myocardial infarction may be detected and appropriately treated to forestall the acute episode. About 25 per cent of patients who are victims of sudden death from coronary artery disease had sought medical advice during the week before death without being detected inspite of laboratory studies such as electrocardiogram and enzyme reports (Kuller, 1969). The insistence of the medical profession on the presence of ischaemic pain and electrocardiographic and enzyme change for the diagnosis of acute myocardial infarction amounts to waiting until the disease process has progressed too far. That pain is not essential characteristic of acute myocardial infarction is witnessed by its absence in 23 per cent of patients detected by routine electrocardiography to have suffered myocardial infarction during the preceding two years. Such painless infarcts are more likely in patients with prior diabetes mellitus or hypertension (Margois, 1973). The absenrce of anginal pain in such patients may be due to decreased pain sensitivity or denial.

To summarise, it may be advisable to respect the patient complaining of *fatigue and weakness* and not just dismiss it as due to over work, anxiety or depression. In the absence of a well defined cause, fatigue and weakness should be considered ominous predictor of prospective myocardial infarction especially in coronary prone patient. Similarly, *indigestion* which is unexplained by a reasonable cause needs care and

caution. Abnormal tendency to *sleep*, *irritability*, *exertional dyspnoea*, *PALPITATION*, *paraesthesias* in the arms and *chest pain*, however vague, should receive proper investigation and care. If ventricular gallop is present, it would reinforce the clinical suspicion. Resting and properly supervised exercise electrocardiograms would be helpful if positive. Serum lipid, and blood sugar tolerance should be determined.

Therapeutic Interventions

Any of the clinical expressions, especially in a coronary prone patient, demand therapeutic intervention.

1. *Complete physical and mental rest:* Considering the disabling and fatal potential of myocardial infarction, it is worth while to have two to three weeks physical and mental rest. This may help by promoting collateral circulation and facilitating dissolution or recanalisation of early minor thrombi.

2. *Behavioral theraphy:* These patients should be indoctrinated in the bliss-ful role of leisure and recreation. Baneful influence of work addiction should be emphasised. The practice of religion, faith in God and active but peaceful way of living may go a long way in combating the epidemic onslaught of coronary artery disease afflicting the materialistic world of today which has forsaken religion and virtous way of life. The West is now patronising transcendental meditation introduced by Maharishi Maresh Yogi. Hans Selye, the protagonist of stress therapies, in his Foreword to a book "Discovering Inner Energy and Overcoming Stress", remarks that transcendental meditation (T.M.) can help humanity face the crisis of modern life. TN is infact a poor form of MARAQABA as practiced in Islam. The way of Islam is the normal and natural way to save humanity from the present day crisis provoked by stress which is an important determinant of final fateful event on the canvass of coronary atherosclerosis.

3. *Treatment of associated conditions:* Associated conditions such as hypertension, diabetes mellitus, hyperlipidaemia should receive appropriate treatment.

4. Avoid smoking, tea ,coffee.

5. Exercise training programmes appropriate to the patient's age, sex and physical condition after the intial period of rest would give a feeling of general well-being, improve the patient's response to exercise without unduly causing tachycardia and rise in blood pressure.

6. Tranquilizers may be needed to relieve tension and anxiety.

7. Propranolol may be helpful by decreasing the myocardial oxygen consumption.

8. Aspirin or dipyridamole may perhaps help in such patients by preventing platelet adhesiveness.

Considering the high incidence of sudden deaths, staggering prehospital mortality, still high coronary care unit death rate and high incidence of morbidity in survivors of acute myocardial infarction, it is perhaps appropriate to study further the prodromes of acute myocardial infarctin so that therapeutic interventions at the earliest may help salvage a substantial percentage of prospective candidates for myocardial infaiction.

References :

1. Moss, A.J. and Goldstein, S: Arch. Int. Med. 129:713, 1972.
2. Lukl, P: in Russek, H.L. and Zohman, B.L. (eds) Coronary Heart Disease; Lippincott: Philadelphia, 1971; p. 270.
3. Kulber, L: Amer. J. Card; 24:617, 1969.
4. Fulton, M: Jubian, D.G. Oliver, M.F. Circulation 40:182, 1969.

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