

# PREMATURE MYOCARDIAL INFARCTION IN SINGAPORE - RISK FACTOR ANALYSIS AND CLINICAL FEATURES

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## ABSTRACT

*A retrospective analysis was carried out on all patients aged 40 years and below with acute myocardial infarction admitted to the coronary care unit (CCU) at the National University Hospital (NUH). We found that most came early direct to hospital, more than half of those eligible received thrombolysis and there was a low incidence of complications. Compared to the Singapore population, Indians in the group were over-represented. The most common coronary risk factor was smoking. A majority had elevated serum cholesterol level. These observations may provide some insight for the primary prevention of premature coronary artery disease in young people in Singapore.*

**Keywords:** myocardial infarction, young coronary risk factors, overseas Indians.

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## INTRODUCTION

Acute myocardial infarction is a leading cause of mortality in Singapore. Consequently, there is much interest in primary and secondary coronary artery disease prevention. This is especially important for Singapore's relatively young population. Our study examines clinical features and risk factors in young Singaporeans who sustained acute myocardial infarctions.

## SUBJECTS AND METHODS

### Patient selection

National University Hospital is a national tertiary referral centre as well as a general hospital covering the south-western sector of Singapore. Our study spans a 27-month period from January 1990 to March 1992. All patients aged 40 years or less admitted to the CCU with proven myocardial infarction were identified and their clinical records reviewed in detail by a single reviewer. From local epidemiological data, there is a steep rise in cardiovascular mortality<sup>(1)</sup> only after the age of forty which was thus used as the cut-off age for "young" patients.

The diagnosis of myocardial infarction was based on the presence of at least two of the following criteria:

- (1) chest pain typical of myocardial infarction with its associated features,
- (2) electrocardiographic (ECG) abnormalities typical of myocardial infarction,
- (3) acute rise in "cardiac" enzymes - total creatinine kinase (CK) and the CK-MB subfraction - to more than twice our laboratory's reference range.

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## Collection of data

Data collected included age, gender, ethnic origin, duration of hospitalisation, nature and duration of symptoms, presence and extent of known major coronary risk factors (diabetes mellitus, hypertension, dyslipidaemia, smoking and a positive family history) and clinical outcome.

Diabetes mellitus and hypertension were recorded if a prior or current diagnosis had been made using standard criteria. Hyperlipidaemia was diagnosed if the fasting serum total cholesterol level obtained within the initial admission exceeded 6 mmol/L. Smoking was recorded if the patient was either currently smoking or had smoked previously.

The follow-up period ranged from 2 to 29 months.

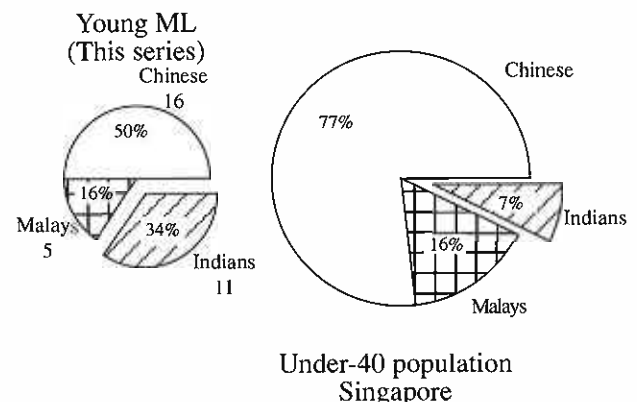
All data points were entered in a computerised database and analysed using standard statistical measures. Data are expressed as mean  $\pm$  1 standard deviation. Significance was established at  $p < 0.05$ .

## RESULTS

### Demography

During the study period, 32 patients met the criteria for inclusion. Their ages ranged from 32 to 40 years ( $37.5 \pm 2.4$ ). All were males. There were 11 Indians (34%), 16 Chinese (50%) and 5 Malays (16%). The distribution was 7.1% Indians, 77.7% Chinese and 14.1% Malays in the general population and 7.0% Indians, 76.0% Chinese and 16.0% Malays in the under-40 population<sup>(2)</sup> (Fig 1).

Fig 1 - Ethnic distribution



## Presentation

Patients arrived in hospital at an average of 4.2 ( $\pm 4.3$ ) hours after the onset of symptoms (range 1 to 24 hours). Eighty-eight percent (28/32) came directly to hospital instead of through a general practitioner. All but one presented with severe gripping chest pain. Associated symptoms included sweating (56%), nausea and vomiting (31%), dyspnea (22%) and giddiness (9%). Other main clinical features are summarised in Table I.

**Table I – Clinical features**

Features	Number (n=32)	Percentage
Severe chest pain at presentation*	31	97
History of prior angina	15	47
Left ventricular failure	4	13
Complex ventricular arrhythmias	9	28
Cardiogenic shock	4	13

Note: n = 32. Each patient may have > 1 clinical features

\*Severe chest pain was associated with other symptoms as detailed in the text

Sixteen patients had anterior myocardial infarction, 12 had inferior, 3 developed infarct at the lateral wall, while one had ECG changes involving more than one territory. Twenty-three of the 32 patients had ST elevation myocardial infarction. Of these, only thirteen patients (57%) received thrombolytic therapy. Two patients were eligible, but did not receive thrombolytic therapy because of delayed diagnosis. Two died before lytic therapy could be given, 5 presented too late for thrombolysis, one had active peptic ulcer disease. The 9 patients with ST depression infarction were not thrombolysed. Streptokinase was used in 10 and recombinant tissue plasminogen activator in the remaining three patients.

The mean maximum CK was  $3129 \pm 2739$  with a mean maximum CK-MB of  $154 \pm 138$ . The time to reach this maximum was  $15 \pm 7$  hours from the onset of symptoms (serial determinations taken at eight hourly intervals).

Twenty-nine of the 30 survivors were studied angiographically. All had evidence of atherosclerosis - 10 had multivessel disease, 14 single vessel disease and 5 had minor coronary artery disease. Infarction related artery (IRA) was identified with the help of q waves or ST changes on the ECG, and wall motion abnormality on the left ventriculogram if performed. The LAD was the IRA in 13 patients, the RCA in 14 patients, while 2 patients had circumflex artery as the IRA.

## Risk factors

Six of the 32 (19%) were hypertensive; 5 of them were on medications. Of the 31 whose smoking status was known, 25 were current smokers and one an ex-smoker. They smoked  $21.6 \pm 14.2$  cigarettes for  $15.8 \pm 5.1$  years. Six patients (19%) had had a lipid profile prior to their infarction. Two were told that their lipid status was "normal" while the other 4 were on lipid-lowering drugs.

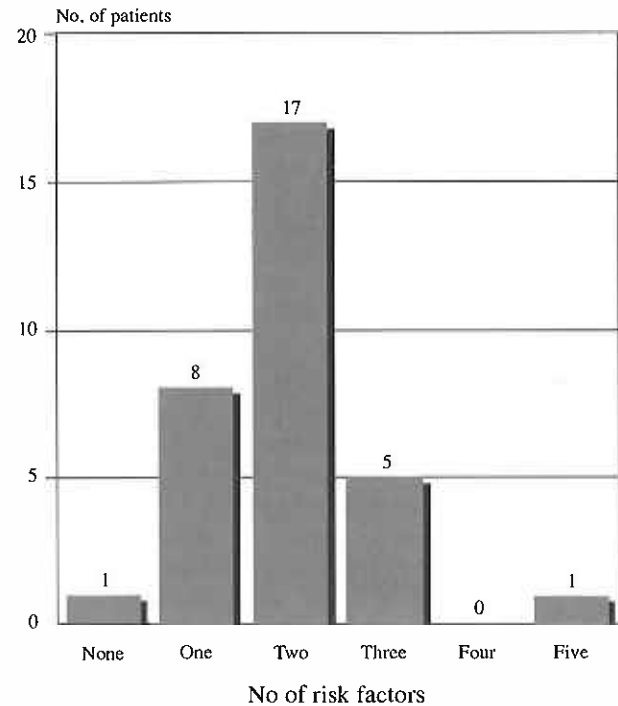
The lipid profile was done for 27/32 patients (84%) at  $42.8 \pm 60.9$  hours after admission. The mean total cholesterol was  $6.24 \pm 1.07$  mmol/l, and 15 (56%) had levels above 6.2 mmol/L. The mean serum triglyceride level was  $2.15 \pm 0.89$  mmol/L. Of the 24 patients with measurements, 10 had HDL cholesterol levels below 0.9 mmol/L, while 20 had LDL cholesterol values above 3.4 mmol/L.

Diabetes mellitus was present in 5 (16%) and 4 of them were on medications. Excluding 2 patients who died shortly after admission, 6 of the remaining 30 patients had a family

history of myocardial infarction in a first degree relative. Five patients had other medical conditions, mainly peptic ulcer disease or "gastritis". None had a history of cerebrovascular disease.

The 32 young men had a total of 48 major coronary risk factors giving a mean of 1.5 risk factors (range 2-5) per patient. Their frequency and distribution are shown in Fig 2 and Table II.

**Fig 2 – Risk factors distribution**



**Table II – Frequency of coronary risk factors**

Risk factors	Number	Percentage %
Hypertension	6/32	19
Family history	6/30*	20
Smoking	26/31*	84
Diabetes mellitus	5/32	16
Hypercholesterolaemia	15/27**	56

\* A family history could not be obtained from 2 of the patients who died soon after admission; 1 of these also did not give a smoking history.

\*\* Five patients did not have a lipid profile.

There were two early hospital deaths, both within 24 hours of admission. One was found at post mortem to have severe triple vessel disease, the other was a Muslim and his family rejected the request for an academic post mortem. They had at least one risk factor each.

## DISCUSSION

To our knowledge, this is the first published report describing the presentation and risk factors for myocardial infarction in the young in Singapore. A similar series in an Asian community was recently reported by Rajadurai et al<sup>(3)</sup> from the University Hospital in Kuala Lumpur.

We observed a disproportionate number of ethnic Indians in our study population. Overseas Indians have been shown to have a higher incidence of coronary artery disease not only in their country of origin, but also the country of adoption<sup>(4)</sup>. This has been noted in Singapore since Muir's large

pathological survey thirty years ago<sup>(5)</sup>.

There is no ready explanation for the susceptibility among Indians to coronary disease. In her study, Rajadurai found elevated serum triglyceride levels in the Indians. In our study, the serum triglyceride in Indians ( $2.44 \pm 0.81$  mmol/L) and non-Indians ( $2.03 \pm 0.91$  mmol/L) were not significantly different. A much larger population-based study<sup>(6)</sup> has also found no significant difference between the fasting serum triglyceride levels of young Indians and non-Indians ( $1.62$  vs  $1.51$  mmol/L).

Compared to the Malaysian series, we observed a higher proportion of smokers (84% versus 57%), but a similar incidence of hypertension and diabetes mellitus. Levels of mean serum cholesterol and triglycerides were comparable<sup>(7)</sup>. The prevalence of smoking in the 30-40 years age group in Singapore is around 20% compared to 25% a decade ago<sup>(8)</sup>. A positive family history has been reported as common in one large series<sup>(9)</sup> but was only present in 20% of our patients. This is similar to the Malaysian figure. Although familial clustering of dyslipidaemia and smoking are recognised, multivariate analysis has indicated that a positive family history is an independent risk factor for coronary artery disease<sup>(10)</sup>.

Some interesting observations can be made by comparing our present series with the small series which was part of Dolder and Oliver's study<sup>(11)</sup> almost 20 years ago. Then, Singapore (and Bombay) were distinguished from the other centres (Auckland, Cape Town, Edinburgh, Heidelberg, Melbourne, Tel Aviv, Los Angeles and Atlanta) by less dyslipidaemia, but more smoking. Singaporean levels of diabetes mellitus and hypertension were comparable.

Since then, the average number of risk factors has risen marginally (1.5 versus 1.33), because a slight decline in smoking has been more than offset by undesirable changes in serum lipid levels. Then, 50% had total serum cholesterol values of  $<5.18$  mmol/L and 64% had serum triglycerides of  $<1.34$  mmol/L. Now, in our series, 81% had serum cholesterol  $>5.20$  mmol/L and 70% had serum triglycerides  $>1.34$  mmol/L. The lipid profile of Singaporeans<sup>(12)</sup> is similar to, and possibly worse than, that of many "developed" nations.

Females were absent from our series. The age-standardised male to female relative risk for mortality from ischaemic heart disease ranges from 4.0 to 11.4 for the 30-39 years age group<sup>(13)</sup>. Myocardial infarction is distinctly rare in pre-menopausal females.

Most of our patients came direct to hospital early. This probably reflects better education and health awareness among our younger population. As a consequence, almost half received thrombolytic therapy. As younger patients are more likely to be physically and economically active, maximum myocardial preservation is important.

Virtually all the survivors in our series were studied angiographically. The high proportion of single vessel disease observed in young infarct patients has previously been reported<sup>(14)</sup>. Coronary arteriography is recommended for

survivors because of the low predictive value of a negative stress test for significant (especially single vessel) coronary artery disease in young patients<sup>(15)</sup>. It is useful for determining prognosis and guiding therapeutic choices<sup>(16)</sup>.

Young infarct patients have less extensive disease than their older counterparts. Like their elders in Singapore and elsewhere, young Indian males are more prone to infarction. Dyslipidaemia and cigarette smoking are particularly prevalent modifiable risk factors in the young. As their disease is less extensive, secondary prevention is vital for preventing disease progression and, possibly, inducing its regression.

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