SUBENDOCARDIAL VERSUS TRANSMURAL MYOCARDIAL INFARCTION: CLINICAL COMPARISON AND REVIEW

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SYNOPSIS

A prospective study was done on 304 consecutive patients admitted to Tan Tock Seng Hospital with confirmed diagnosis of acute myocardial infarction using the WHO criteria. The patients were divided into two groups, ie 218 patients with transmural myocardial infarction, and 86 patients (28.3%) with subendocardial infarction. The clinical characteristics were compared. The epidemiological characteristics of age and ethnic group of the two grops were comparable. The occurence of complications, except for atrial fibrillation, right bundle branch block and pericarditis, were not statistically different in the two groups. There was a significantly higher percentage of patients with cardiomegaly in the sub-endocardial infarction group (79%) as compared to that in the transmural infarction group (61.9%) (p 0.01).

The in-hospital mortality of 10.5% in the subendocardial infarction group was lower than the 21.1% in the group with infarction ($p < 0.05$).

INTRODUCTION

The subendocardium is the most vulnerable part of the myocardium. Several reviews and reports on the anatomy, haemodynamics, physiological characteristics and electrophysiological properties of the subendocardium are available in the literature (1-10, 27-29). Clinical reports (11-19, 23, 36, 37, 39-41) on subendocardial infarction are by far fewer in number than the mass of clinical studies on almost every aspect of transmural myocardial infarction. The reason for this may be the difficulty in making a clinical diagnosis of subendocardial infarction. One has to rely on the combination of clinical criteria and investigations for making the diagnosis (11). In this report, we compared the clinical characteristics of the two groups of patients i.e. the transmural and subendocardial infarction groups.

MATERIALS AND METHODS

This study was a prospective study of 304 consecutive admissions into Tan Tock Seng Hospital between October 1979 to October 1980. The study population was unselected and included all patients admitted to Tan Tock Seng Hospital with a confirmed diagnosis of actue myocardial infarction using the following three W.H.O criteria:-

1) Compatible chest pain of more than twenty minutes in duration.
2) Typical electrocardiographic pattern of myocardial infarction documented serially on standard 12 lead electrocardiograms.
3) Typical pattern of elevation of serum cardiac enzymes.

The population under study was not confined to patients admitted into the intensive care area. All patients who were admitted into the Tan Tock Seng Hospital, regardless of the state on admission (moribund patients included), were accepted into the study.

Standard 12 lead electrocardiograms were obtained daily, for the first three days, and sometimes more than once a day, if required.

The electrocardiographic criteria for transmural myocardial infarction were:-

1) Q wave duration equal to or more than 0.04 seconds.
2) Q wave amplitude equal to or more than 25% of the amplitude of R waves in the same lead. This should be accompanied by initial ST segment elevation and followed temporarily by T wave inversion.

The electrocardiographic criteria for subendocardial infarction were:-

a) ST segment depression of the ischaemic "square wave" type in the limb and/or precordial leads and lasting for at least 48 hours and/or;
b) Deep and symmetrical new T wave inversions in some or all precordial leads which occurred gradually and persising for days or weeks;
c) Absence of pathological Q waves;
d) ST segment elevation in lead AVR. This may not be present in all cases of subendocardial infarction.

The Chi-square method and the Student's test were used for statistical analysis. Propability values of less than 0.05 were considered significant.

RESULTS

There were 304 patients in the study population. Of these, 218 patients satisfied the criteria for transmural myocardial infarction and 86 patients satisfied the criteria for subendocardial infarction.

Sex:

There were 182 males and 36 females in the transmural group and 60 males and 26 feamles in the subendocardial group. The male to female ratio was 5:1 in the transmural group. The male predominance was present but to a lesser degree in the subendocardial group in which the male to female ratio was 2.5:1.

Age:

The mean age of the patients in the transmural group was 59.2 years, and that of the subendocardial group was 60.3 years. The distribution of the various age groups was comparable in the two groups. The peak incidence was in the sixth decade in both groups. Details are seen in Table 1.

Ethnic Group Distribution:

There was no statistical difference in the distribution of ethnic groups in both groups of patients. (Table 2)

TABLE 1: AGE DISTRIBUTION

Age Group (years)	Transmural Infarction		Subendocardial Infarction	
	Number	Percentage	Number	Percentage
20 — 29	1	0.4	0	0
30 — 39	8	3.7	2	2.3
40 — 49	32	14.7	11	12.8
50 — 59	76	34.9	33	38.4
60 — 69	69	31.7	24	27.9
70 — 79	23	10.5	11	12.8
80 — 89	9	4.1	5	5.8
	218	100.0	86	100.00

$X^2 = 1.86$ $p < 0.05$

TABLE 2: ETHNIC GROUP DISTRIBUTION

Ethnic Groups	Transmural Infarction		Subendocardial Infarction	
	Number	Percentage	Number	Percentage
Chinese	118	54.1	52	60.5
Malay	35	16.1	17	19.7
Indian	55	25.2	16	18.6
Other races	10	4.6	1	1.2
Total	218	100.0	86	100.0

$X^2 = 4.10$ $p > 0.05$

Physical Activity:

A history of the level of physical activity six months before infarction was available in 186 patients in the transmural group and 78 patients in the subendocardial group. 19.4% of the transmural group and 18% of the subendocardial group had done some form of exercise. The difference was not statistically significant. (Table 3)

Risk Factors:

A history of smoking was found in 37.6% of the transmural group and 45.3% of the subendocardial group. Hypertension was present in 32.6% of the subendocardial tension was present in 32.6% of the transmural group and 39.5% of the subendocardial group. Diabetes mellitus was present in 34.9% of the transmural group and 27.9% of the subendocardial group. Hyperlipidaemia was present in 2.3% and 5.8% of the transmural and subendocardial groups respectively. Family history of ischaemic heart disease was present in 8.7% of the transmural and 9.3% of the subendocardial groups. The differences in the presence of each of the risk factors in the two groups was statistically insignificant. (Table 4)

TABLE 3: PHYSICAL ACTIVITY DURING THE SIX MONTHS PRIOR TO INFARCTION

Physical Activity	Transmural Infarction		Subendocardial Infarction	
	Number	Percentage	Number	Percentage
Exercise at least 3 times per week	21	11.3	12	15.4
Exercises Occasionally	15	8.1	2	2.6
No Exercise	150	80.6	64	82.0
TOTAL (KNOWN)	186	100.0	78	100.0

$X^2 = 3.33$ $P > 0.05$

TABLE 4: RISK FACTORS

Risk Factors	Transmural Infarction		Subendocardial Infarction		
	Number with Positive Risk Factors	Percentage of a Total of 218	Number with Positive Risk Factors	Percentage of a Total of 218	X^2
1. Smoking	82	37.6	39	45.3	1.27 $p > 0.05$
2. Hypertension	71	32.6	34	39.5	1.03 $p > 0.05$
3. Diabetes Mellitus	76	34.9	24	27.9	1.05 $p > 0.05$
4. Hyperlipidaemia	5	2.3	5	5.8	1.42 $p > 0.05$
5. Family History	19	8.7	8	9.3	0 $p > 0.05$

Preceding History of Angina and Myocardial Infarct:

A history of previous angina was present in 104 patients (47.7%) in the transmural group, and 30 patients (34.8%) in the subendocardial group. Chi-square analysis showed this difference to be statistically insigificant. (Table 5)

A history of documented prior infarction was present in 20 patients (9.4%) in the transmural group and 14 patients (16.4%) in the subendocardial group. The difference was not statistically significant. (Table 6)

Presenting Symptom:

Typical chest pain was the presenting symptom in 192 patients (88.1%) in the transmural group and in 66 patients (76.7%) in the subendocardial group ($P < 0.05$). The rest of the patients in both groups presented with either atypical chest pain, no chest pain, syncope or breathlessness. (Table 4) A significantly higher percentage of patients with transmural myocardial infarction presented with typical chest pain. (Table 7)

TABLE 5: HISTORY OF PRECEDING ANGINA

History of Preceding Angina	Transmural Infarction		Subendocardial Infarction	
	Number	Percentage of total	Number	Percentage of total
No	114	52.3	56	65.2
Yes	104	47.7	30	34.8
Total	218	100.0	86	100.0

$X^2 = 3.61$ $P > 0.05$

TABLE 6: HISTORY OF PREVIOUS MYOCARDIAL INFARCTION

History of Previous Infarction	Transmural Infarction		Subendocardial Infarction	
	Number	Percentage of total (known)	Number	Percentage of total (Unknown)
No Previous Infarct	193	90.6	71	83.6
Previous Infarct	20	9.4	14	16.4
Total Known	213	100.0	85	100.0

$X^2 = 1.44$ $P > 0.05$

TABLE 7: PRESENTING SYMPTOMS

	Transmural Infarction		Subendocardial Infarction		
	Number	Percentage of Total	Number	Percentage of Total	X^2
Typical Chest Pain	192	88.1	66	76.7	5.31 $p < 0.05$
Atypical Chest Pain	10	4.6	5	5.8	0.02 $p > 0.05$
No Chest Pain Asymptomatic	9	4.1	9	10.5	3.38 $p > 0.05$
With Syncope	2	0.9	2	2.3	0.17 $p > 0.05$
With Breath-lessness	5	2.3	4	4.7	15.1 $p > 0.05$
TOTAL	218	100.0	86	100.0	

Dysrhythmias:

The occurrence of the various major dysrhythmias during the hospital stay is as shown in Table 8. The occurrence of dysrhythmias in both transmural and subendocardial group was not statistically difference except for the higher incidence of atrial fibrillation in the subendocardial group, ($p < 0.05$) and right bundle branch block in the transmural group ($p < 0.01$).

It would appear that patients with subendocardial infarction are just as susceptible to the major dysrhythmias and heart blocks as those with transmural infarction.

Systemic Embolism:

Systemic embolism was a complication in 8 patients (3.7%) in the transmural group in 2 patients (2.3%) in the subendocardial group.

Pericarditis:

Pericarditis occurred early during the first week of infarction in 21 patients (9.6%) in the transmural group but there was none in the subendocardial group ($P < 0.01$).

Radiographic Cardiomegaly:

Radiographic evidence of cardiomegaly was defined as cardiothoracic ratio of greater than 0.5. A significantly higher percentage 79% of patients with subendocardial infarction had cardiomegaly compared to 61.9% in the transmural group ($P < 0.01$).

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Severity:

The severity of infarction was classified into three groups as follows:-

1) Patients with cardiac failure.
2) Patients without cardiac failure.
3) Patients with cardiogenic shock.

TABLE 8: DYSRHYTHMIAS

Dysrhythmias	Transmural Infarction		Subendocardial Infarction		X^2 Text
	Number	Percentage of Total	Number	Percentage of Total	
1. Atrial Fibrillation	5	2.3	8	9.3	5.79 $p < 0.05$
2. Ventricular Febrillation	8	3.7	0	0	1.97 $p > 0.05$
3. Ventricular Tachycardia	9	4.1	2	2.3	0.17 $p > 0.05$
4. 1° Atrio-Ventricular Block	10	4.6	1	1.2	1.21 $p > 0.05$
5. Mobitz Type 1 Block	8	3.7	1	1.2	0.62 $p > 0.05$
6. Mobitz Type 2 Block	4	1.8	0	0	0.05 $p > 0.05$
7. 3° Atrio-Ventricular Block	15	6.9	1	1.2	2.98 $p > 0.05$
8. Right Bundle Branch Block	26	11.9	0	0	9.74 $p < 0.01$
9. Left Bundle Branch Block	9	4.1	4	4.7	0 $p > 0.05$
10. Left Anterior Hemiblock	17	7.8	3	3.5	1.23 $p > 0.05$
11. Left Posterior Hemiblock	2	1.0	0	0	0.01 $p > 0.05$
12. Bifascicular Block	6	2.8	0	0	1.20 $p > 0.05$
13. Trifascicular Block	2	1.0	0	0	0.01 $p > 0.05$
TOTAL	121		20		

TABLE 9: SEVERITY

Subject	Transmural Infarction		Subendocardial Infarction	
	Number	Percentage of Total	Number	Percentage of Total
No Cardiac Failure	133	61.0	51	59.3
Cardiac Failure	70	32.1	32	37.2
Cardiogenic Shock	15	6.9	3	3.5
TOTAL	218	100.0	86	100.0

DISCUSSION

The innermost muscle layer of the left ventricle, the subendocardium, is the most vulnerable layer to ischaemia and infarction. (1,2). The subendocardium is poorly perfused at a time when its needs are greatest as during systole. The intramyocardial compressive forces are greatest in the subendocardium and increasing coronary vascular resistance during systole may lead to virtual absence of flow to the deeper layers. The subendocardium is hence dependent upon adequate diastolic perfusion to maintain its oxygen supply. The muscle fibres of the subendocardium are the largest in the ventricular wall. Electron microscopic studies have shown that subendocardial sarcomeres are longer in diastole and shorter in systole than the superficial fibres. These two factors, the greater dependence of the subendocardium on diastolic coronary perfusion and its greater energy requirements account for the vulnerability of the subendocardial layer to ischaemia and infarction.

The clinical diagnosis of subendocardial infarction remains difficult. There is as yetno uniform anatomical definition of the thickness of the subendocardial layer of the myocardium. (3). In clinicopathological correlations, (4, 7, 20) the inner third of the myocardium has been defined as the subendocardial layer. The electrocardiographic features of ST segment depression, T wave inversion and ST elevation in lead AVR with these lesions have been described. (4, 7, 20). Others, who have included a greater thickness of myocardium in the infarcted tissue, have described loss of R waves or deep Q waves as well as ST segment and T wave changes. (3, 4, 6, 20). In the absence of a new definite laboratory test, the diagnosis of subendocardial infarction depends on a combination of a compatible history, electrocardiographic changes and elevation of serum cardiac enzymes.

Transmural myocardial infarction is usually attributed to acute coronary occlusion, often by fresh local arterial thrombus superimposed on an old atheromatous plaque. (10). There are reports that myocardial infarction may occur with normal coronary arteries as demonstrated by subsequent coronary arteriograms. A subendocardial infarction however is often not due to acute thrombotic occlusive events. (10). Subendocardial infarction often occurs in the setting of reduced coronary perfusion pressure. This may occur in systemic hypotension from any cause, decreased collateral flow or sudden increases in coronary obstruction due to subintimal haemorrhage. Subendocardial necrosis may occur in left ventricular outflow tract obstruction, left heart failure without atherosclerosis, severe aortic regurgitation, pulmonary embolism, subarachnoid haemorrhage, pheochromocytoma and restrictive pericarditis.(1).

The present study shows that the two groups of transmural and subendocardial infarction were comparable in the epidemiological characteristics namely, age distribution, ethnic group, level of physical activity six months prior to infarction and the presence of risk factors.

Males predominated in both groups, but there were more females in the subendocardial group than in the transmural group. The male to female ration was 5:1 in the transmural group and 2.5:1 in the subendocardial group. Similar findings have been shown in the study of Madias et al. (12).

A history of previous myocardial infarction was present in 16.4% of the subendocardial group and 9.4% of the transmural group (statistically not significant). The study of Schneinman and Abbott (16) reported a history of infarction 31% of the non-transmural group. The study by Madigan et al (13) reported a previous myocardial infarction in 24% of the subendocardial group.

A history of preceding angina was present in 48% of this series of patients with transmural myocardial infarction and in 35% of patients with subendocardial infarction (the difference was not statistically significant). Sheinmann and Abbott (16) reported a history of angina in 38% of patients with non-transmural infarction and 39% in the transmural group.

Madigan et al (13) reported a higher percentage (80%) of prior angina in the subendocardial group. The differences in the various studies could have been due to patient selection. The last study for example, included only patients who had undergone coronary angiographic studies.

The severity of infarction was divided into three subgroups ie. no cardiac failure, cardiac failure and cardiogenic shock. This present study showed that the two groups of patients with transmural and subendocardial infarction were comparable in terms of severity. Similar findings were found in other studies. (13 — 14)

With the exception of atrial fibrillation and right bundle branch block, there was no statistically significant differences in the occurrence of major dysrhythmias and heart blocks in both transmural and subendocardial groups of patients. The complication of major dysrhythmias has been shown to be comparable in both sub-

endocardial and transmural myocardial infarction. (12 — 15)

The occurrence of major heart blocks, with the exception of right bundle branch block was not statistically different in both the groups. However, there was a higher incidence of right bundle branch block in the transmural group and none in the subendocardial group (P 0.01).

The initial chest radiograph in 61.9% of patients in the transmural groups showed cardiomegaly (defined as cardiothoracic ratio of more than 0.5). A greater proportion (79%) of patients in the subendocardial group presented with radiographic cardiomegaly on the first day of admission. The reasons for this may be explained on a physiological basis. The precardios balance of blood supply to the subendocardium in dilated or hypertrophied hearts from whatever cause, will result in a greater propensity for subendocardial infarction in these hearts when compared to a normal sized heart.

The incidence of systemic embolism small as it may be, was not statistically different in the two groups. This is not unexpected as the subendocardial infarcted area is a common denominator in both groups. The only difference is that the transmural group has a full thickness infarct. The subendocardium is the area where small thrombus may form and be a source of systemic embolism.

Pericarditis early in the course of infarction occurred in 9.6% of the patients in the transmural group. None of the patients in the subendocardial group were found to have pericarditis. Pericarditis early in the course of infarction, in contrast to the Dressler's syndrome, is due to involvement of the pericardium by the full thickness infarct in transmural infarction which is not expected to occur in subendocardial infarction.

Post-infarction angina was present in comparable proportion in both groups of patients, 32% in the transmural group and 29% in the subendocardial group. This was comparable to the incidence of post-infarction angina reported by Fabricius-Bjerre et al. (19). Their study however was on the incidence in patients who survived infarction and who had angina pectoris at the end of 5 years. Madigan et al (13) reported a higher percentage of patients with angina pectoris after subendocardial infarction. They reported, that after a mean follow up of 10.6 months, 30% of patients had stable angina and 40% had unstable angina.

The early in-hospital mortality in the subendocardial infarction group in this study was 10.5% and 21.1% in the transmural group (difference is statistically significant P 0.05). This lower mortality in patients with subendocardial infarction was in agreement with other reports (3, 7, 12, 16, 17, 18, 23, 36, 37, 39, 47). No differences in mortality between the transmural and non-transmural groups were found in a few reports (12, 14).

Thanavaro et al (37) further divided the patients into subgroups according to peak serum glutamic oxaloacetic transminase levels, and found a correlation between hospital mortality and peak serum glutamic exaloacetic transaminase levels.

The long term prognosis of patients who survived subendocardial infarction has been compared to that of patients who survived transmural myocardial infarction. Cannom et al (15) followed the survivors of transmural (Group I) and non-transmural infarction (Group 2) for 36 months. Their study found a high incidence of sudden death after discharge ie. 33% in subendocardial infarction group versus 15% in transmural group (P 0.02). There was a higher incidence of death from all cardiac causes (41.6% in Group 2 versus 24.3% in Group 1, P 0.05). Their report and that of Smeets et al (47) suggest that patients with non-transmural myocardial infarction have a guarded prognosis.

Fabricius Bjerre et al (19) in a five year survival study of patients who survived subendocardial and transmural myocardial infarction found that the five year survival rates were not statistically different in the transmural infarction and subendocardial infarction group, 59% and 57% respectively. Other reports (14, 39, 41) have shown similar long-term prognosis in survivors of both groups of patients.

Geltman et al (40) found a lower mortality of 6% in subendocardial infarction group and 16% in transmural group of patients at the end of an average follow-up period of 21.7 months. However, when patients were grouped according to Infarct Size Index (ISI) enzymatically, those with small infarcts (15 CK-g-eq/metre^2) had a signficantly better survival rate, than those with large infarct size. This was regardless of whether the infarct was transmural or subendocardial. These results indicate that the extent of infarction is a better determinant of prognosis than its nature of distribution in the left ventricle.

Coronary thrombosis was found in 10% patients who died suddenly or in whom necrosis is limited to the left ventricular subendocardium. (10). A greater proportion (50%) of patients with transmural infarction were found to have coronary thrombus. (10). Levine and Ford (20) in their study, found a common pathologic denominator in the patients with subendocardial infarction. They suggested that it was the deficient irrigation of the entire coronary arterial system, and not of a single coronary artery, that was necessary for the production of subendocardial infarction. This concept was supported by subsequent clinical reports in which coronary arteriograms were done to delineate the coronary arterial tree. The prospective study from the Mayo Clinic (13) delineated the coronary system in all 50 patients with subendocardial infarction. Coronary arteriography demonstrated significant lesions defined as more than 75% narrowing in at least one vessel in all 50 patients. Single vessel disease was found in 20 patients or 40 percent of patients. Sixteen patients or 32% had double vessel disease and fourteen patients or 28% had triple vessel disease. Schnulze et al (32) found no difference in the extent and severity of coronary artery disease when they compared the results of coronary angiography and left ventriculography in survivors of transmural and non-transmural myocardial infarction.

This finding is not surprising, as the syndromes of angina, unstable angina, subendocardial infarction, transmural myocardial infarction are a continuing spectrum of disease the basis of which is an imablance between the demand and supply of vital needs to the myocardium. It is possible for a patient to present with a whole spectrum of syndromes in order and culminating in a full thickness transmural infarction. It is also possible for a patient to present initially as transmural infarction without any preceding angina or subendocardial infarction.

It must be stressed that a clinical diagnosis of transmural myocardial infarction is difficult, (11, 21, 38)

and is even more so in the case of subendocardial infarction. Hence the diagnosis must necessarily be made on the combination of several criteria. There is a possibility for subendocardial infarcts in some patients to remain undetected for lack of sufficient criteria to satisfy a definite diagnosis. There is a need for sensitive and specific non-invasive tests to accurately delineate the location and extent of myocardial infarction.

The incidence of subendocardial infarction in this series was 28.3%. This is fairly similar to previous reports of 24.7%, 28.5% and 23% by Lown (43), Meltzer and Kitchell (44) and the Norwegian Multicenter Study Group (45) respectively. However, the figure is much higher than that from a previous report from Singapore of only 9% (46). The low incidence in the previous study could be due to that study being done on patients who were seen before 1976. The disease pattern of ischaemic heart disease in Singapore appears to have changed to that in the Occident.

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