

SILENT MYOCARDIAL ISCHEMIA

Pages with reference to book, From 165 To 167

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Coronary artery disease has been called a modern epidemic because of its prevalence in different parts of the world.¹ In the U.S.A. the prevalence of this disease increased from 7.9 in 1930 to 290, per thousand in 1963 but from 1968 to 1976, there was 25% decrease in its prevalence², which has been attributed to the wide-spread dissemination of knowledge of the risk factors and alteration of life style³. The manifestations of coronary artery disease embrace a wide spectrum from the benign minor coronary atherosclerosis without angina or ischemia to sudden death. Researches in the last few decades led to the concept of prevention, medical management and surgical myocardial revascularization and also to the awareness of asymptomatic coronary artery disease and silent myocardial ischemia. The recognition, pathophysiology, diagnosis, prognosis and therapy of myocardial ischemia is the new clinical challenge.

Silent myocardial ischemia is defined as objective evidence of myocardial ischemia without angina or angina equivalent. The objective evidence consists of ST depression on stress testing, holter monitoring, c.c.u. monitoring, left ventricular wall motion abnormalities, at rest or on exercise, on radionuclide ventriculography or echocardiography, reversible perfusion defect on Thallium scan. Silent myocardial ischemia includes the¹ asymptomatic patients with positive exercise test², silent myocardial infarction, in Framingham study⁴ 30% of myocardial infarcts were silent, diagnosed only by serial electro-cardiography³. The third group of patients with silent ischemia has or had symptomatic ischemic heart disease.

The prevalence of silent ischemic episodes as reported by Schang and Pepine⁵ is three times the symptomatic attacks. The overall rate of silent myocardial ischemia in population is considered to be 2.5%. The magnitude of this problem puts it in its proper perspective and raises some questions. Does silent ischemia precede the dreaded sudden cardiac death? Is silent ischemia harbinger of myocardial infarction? What are the best diagnostic techniques for the evaluation of silent ischemia? Does silent myocardial ischemia require treatment? These are some of the issues calling for urgent answers. Before addressing these issues a brief review of pathophysiology of silent myocardial ischemia is desirable. The work of Schang and Pepine⁵ as mentioned earlier has shown that 75% attacks of myocardial ischemia are silent. They occurred during daily activities as on rising, office work, reading, watching T.V., conversation, driving, etc., most frequent in morning hours following a circadian rhythm. The attacks occurred at a heart rate slower than that evoking depression on exercise testing, unaccompanied by any significant rise in blood pressure. The episodes of silent ischemia may last more than 20 minutes. The stenosed epicardial coronary arteries constrict secondary to ischemia. Abnormal endothelial function predisposes to arteriolar constriction. These are some of the⁶ mechanisms responsible for silent myocardial ischemia.

Silent myocardial ischemia is not as benign as it is believed. Deanfield⁷ has reported silent ischemia could lead to myocardial infarction and to sudden death. According to Washington⁸ study asymptomatic patients with positive E.T.T. have higher risk of future cardiac events. Similar results were reported by Hickman⁹ in coronary angiography proven coronary artery disease. Erikssen¹⁰ study from Oslo showed that asymptomatic patients with coronary artery disease, with positive ETT had 1% mortality per year whereas mildly symptomatic patients with positive ETT had 3% mortality per year. In post myocardial infarction patients' prognosis was better in asymptomatic patients with negative ETT compared to symptomatic patients with positive ETT. This has been confirmed by Theroux¹¹ and

Duke Harvard¹² study. It would appear that silent myocardial ischemia, though not as ominous as symptomatic myocardial ischemia, affects prognosis adversely. It needs evaluation, close supervision and appropriate management, and its evaluation requires new diagnostic techniques, e.g., exercise testing, C. C. U. monitoring, ambulatory monitoring, rest and stress wall motion abnormalities and Thallium scan.

These techniques should be utilised in a cost effective manner. Simple diagnostic procedures like stress testing and ambulatory monitoring should be done first. Ambulatory monitoring provides information about the frequency and duration of myocardial ischemia which has important bearing on selection of patients for further tests and appropriate treatment. Depending on the finding of stress testing and ambulatory monitoring, coronary angiography may be done in selected patients. Interpretation of coronary angiogram in relation to findings of ambulatory monitoring and stress test enables correct decision regarding therapy.

The purpose of therapy is total abolition of all ischemia by measures to decrease the demand and vasoconstrictive element. According to the study of Schang and Pepine⁵ episodes of silent ischemia were reduced by sublingual nitroglycerine, also by transderm nitroglycerine, treatment with nitrites being obviously beneficial.

Beta blockers were used in the treatment of silent ischemia by Gottlieb¹³ They reduced the number and duration of the episodes of silent ischemia. Calcium channel¹⁴ blocking agents have been found useful in denland induced angina but more information is needed for their use in Maseris or mixed angina. In Prinzmetal angina Verapamil¹⁵ reduces the ischemic events by 75%. In view of the platelet abnormalities in patients with ischemic heart disease antiplatelet drugs may seem logical choice though in the management of angina there is only indirect evidence of their benefit. According to Canadian¹⁶ study there was 51% reduction in the risk of cardiac death or myocardial infarction in those taking aspirin. Since silent myocardial ischemia may precede myocardial infarction or sudden death, aspirin by its antiplatelet effect may reduce myocardial ischemia.

Angioplasty¹⁷ offers a new approach in the management of silent ischemia. Proximal lesion in left anterior descending and right coronary artery can easily be dilated and with increased proficiency. At many centres multivessel angioplasty is being undertaken. The success rate of angioplasty is 80-90%, the restenosis rate is 30% in one year. There is 0.2% mortality and about .2- to 3% patients may require emergency bypass surgery.

The role of surgery in silent ischemia needs to be defined. If the guidelines of coronary artery surgery study¹⁸ are followed, only the patients with left main coronary artery and three vessel disease with left ventricular dysfunction should be offered surgery. However, according to European¹⁹ study, patients with three and two vessel disease, with or without left ventricular dysfunction, have better prognosis after surgery.

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