editorial

Asymptomatic Ischaemic Heart Disease

It is a common saying that unless asked for even the mother does not serve milk, but should we apply the same analogy to the patients with ischaemic heart disease (IHD). Should we wait for the ischaemic cry of the heart to draw our attention ? No.

Non-invasive investigations like treadmill test (TMT) ambulatory ECG (Holter) monitoring, exercise radionuclide multigated aquisition (MUGA) study, thallium perfusion scintigraphy and metabolic cardiac imaging (positron emission tomography - PET) have made it possible to unearth total ischaemic burden (symptomatic plus silent ischaemic episodes) [1]. The point to note is that symptomatic ischaemia comprises only tip of iceberg while silent myocardial ischaemia (SMI) continues unabatedly to do the damage as it remains undiagnosed and hence untreated. If untreated, recurrent SMI episodes may end up as acute myocardial infarction (first presentation), ischaemic cardiomyopathy or sudden cardiac death. The SMI has been classified into three subtypes [1,2,3,4].

(i) Asymptomatic patients with angiographically proven coronary artery disease (CAD) and they showed SMI on noninvasive tests as TMT, Holter or MUGA (type I SMI). In general public such patients are difficult to diagnose as it is not feasible to subject all the healthy adult individuals to screening tests like TMT, Holter and MUGA. In contrast persons engaged in high risk professions like flying and those who are being periodically screened to diagnose disease in subclinical state, the detection of SMI is possible.

(ii) SMI in post myocardial infarction (MI) patients with or without symptomatic ischaemia (type II SMI) and

(iii) SMI in patients with chronic stable angina[4,5,6], i.e., in addition to symptomatic episodes (type III SMI).

The patients with Type II and III SMI are certainly the candidates for coronary arteriography to delineate the obstructive anatomy and accordingly decide the revascularisation, either percutaneous transluminal coronary angioplasty (PTCA) or coronary artery bypass graft (CABG) surgery [7,8]

In post MI patients the standard set of investigations done to prognosticate cases (symptomatic or otherwise) are : submaximal TMT (to demonstrate inducible ischaemia), Holter study (to detect arrhythmias) or MUGA / Echo study (to assess left ventricular ejection fraction) and it is concluded that if all the three tests are negative, the patient will have good long term prognosis and depending upon number and severity of positivity of tests, the outcome varies [8].

Coronary angiography is mandatory in post MI patients with :

(i) subjective or objective (TMT/Holter or MUGA) evidence of ischaemia, (ii) successful reperfusion with streptokinase / urokinase or subendocardial (non-q)myocardial infarction, (iii) transient ST-T changes in reciprocal leads during acute myocardial infarction (suggesting multivessel disease) i.e., "ischaemia at distance" and (iv) Young MI patients (less than 40 yrs of age). Further management will depend upon findings of coronary angiography.

In patients with type I SMI we have to individualise therapy according to the coronary obstructive lesions. On long term follow up, large majority of these asymptomatic patients do develop symptoms due to progression of CAD

(existing coronary obstructive lesions increase or new hemodynamically significant lesions appear) and only minority may present as acute MI or end up as sudden cardiac death. However, if anatomy or coronary lesions do not warrant surgery or PTCA, the medical therapy should be optimised and risk factor profile should be stratified to abolish not only symptomatic but also the silent ischaemic episodes in order to improve long term prognosis. This is especially important in patients who reveal SMI of more than 60 min duration in 24 hours as these cases carry poor prognosis.

Why SMI ? The possible causes listed are : (i) inadequate pain stimulus strength because of area of myocardium rendered ischaemic is too small and hence can not muster adequate stimulus to reach perception level, (ii) raised pain threshold even with adequate stimulus strength (iii) autonomic neuropathy as with diabetes mellitus (iv) denervation of heart (post cardiac transplantation), (v) faulty anginal warning system, (vi) excess production of internal opioids like endorphins and metenkephalin and (vii) personality and psychological status [1,2,6]. Some patients tend to underplay their symptoms and ignore early anginal warnings.

That leaves us with the patients who have evidence of ischaemia on TMT/MUGA but angiographically normal epicardial coronary arteries (? false positive TMT) because vessels below 1 mm size are beyond the resolution capacity of coronary arteriography. In these patients first attempt should be made to rule out coronary artery spasm by ergonovine test (to be done in catheterisation laboratory only) and other possibilities like small vessel disease, microvascular angina and deficient coronary vasodilator reserve (syndrome "X") may remain as enigma. Though these patients can technically be labelled to have no CAD, the positive TMT is the strongest risk factor for IHD, more so if it is moderately or strongly positive and associated with other conventional risk factors like hyperlipidemia, smoking or hypertension [9].

Serial follow up over 3-14 years in patients with positive TMT had revealed that risk ratio of developing disease is 6:3 among positive responders as compared to negative TMT and the risk is further skewed up by strongly positive TMT [10].

The percentage of asymptomatic patients with positive TMT having demonstrable significant CAD is around 33 to 50% [11,12,13] with higher incidence among patients with history of angina equivalents, positive family history or other risk factors. In this journal, Singh MM et al have found that significant CAD is observed in 29% of cases with positive TMT and abnormal resting ECG and in 22% of cases with positive TMT and normal resting ECG, thus leaving 71 - 78% asymptomatic patients with positive TMT and normal coronaries. Though these patients are declared to have no CAD they are being periodically evaluated by noninvasive investigations for earliest evidence of deterioration (subjective or objective) and at that juncture, they will need repeat coronary arteriography to rule out or confirm CAD. Thus anginal manifestation in an ischaemic episode is a late feature and patients with recurrent SMI keep stunning the involved segment of myocardium during activiites without the patients being aware of it.

To detect these SMI episodes, apart from PET other noninvasive tests done TMT, Holter, MUGA and Thallium perfusion scintigraphy.

As on now it is recommended that if the TMT/Holter is conclusively evident of SMI in an asymptomatic healthy subject, he should be treated as IHD with SMI. However if only one of the tests is positive for IHD, the patient may be subjected to another noninvasive test (MUGA/Thallium studies) for objective evidence of ischaemia before subjecting him to coronary arteriography, a **"gold standard"** to diagnose CAD.

Some other features of SMI which often occurs during daily activities are [5,6,15] : (i) it is more common than symptomatic ischaemia (ii) it

often precedes the onset of symptomatic ischaemia, (iii) it occurs at lower heart rate as compared to exercise induced ischaemia which occur at higher heart rate meaning thereby that rate pressure produce (multiplication of peak heart rate and systolic BP) does not increase in SMI, (iv) it occurs both at rest and during exercise, (v) it shows diurnal variation with peaks in early morning (6.00 to 8.00 am) and evening, (vi) it may not show any relation to extent of angiographic CAD or left ventricular functions and (vii) medical therapy or PTCA/CABG should abolish all symptomatic and SMI episodes.

In conclusion, patients with SMI with angiographically demonstrable CAD need optimisation of medical therapy or PTCA/CABG depending upon coronary obstructive lesions and patients with asymptomatic SMI with angiographically normal coronaries should be carefully followed up for early detection of CAD as positive TMT is the strongest risk factor for CAD. Post myocardial infarction patients should be actively investigated for SMI/arrhythmia and treated accordingly to improve prognosis.

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