Time To Prognosticate...

Enough has been said about Stable Angina, Unstable Angina, Myocardial Infarction and Angina equivalent symptoms. For the practicing physician, whether allopath or homoeopath, emphasis should be on prognosticating a case of Coronary Artery Disease. Bluntly put, it means trying to assess the chances of complications like Infarction or LVF in a diagnosed case of CAD. To do this would require an in-depth understanding of the risk factors as well as monitoring parameters.

CAD is the commonest cause of Cardio vascular disability and mortality. Male to female overall ratio of those afflicted is 4:1, the ratio is 8:1 before age 40, and it is 1:1 beyond age 70. In men, the peak incidence of clinical manifestations is at age 50-60; and in women at age 60-70.

Risk Factors for Coronary Heart Disease:

A number of important risk factors have been identified for premature coronary heart disease. These include a positive family history (particularly when onset is before age 50), age, male gender, blood lipid abnormalities, hypertension, physical inactivity, cigarette smoking, diabetes mellitus, elevated blood homocysteine levels, and hypoestrogenemia in women. There is overwhelming evidence to show that abnormalities of lipid metabolism play a direct role in the pathophysiology of CAD. We all know that in today's times just asking of Total Cholesterol and Triglycerides would be real foolish as one may have near normal Total Cholesterol with very high LDL Cholesterol. Risk increases progressively with higher levels of LDL cholesterol and declines with higher levels of HDL cholesterol. The ratio of LDL to HDL cholesterol provides a composite marker of risk, with ratios below 3 indicating a lower risk and ratios above 5 indicating higher risk. Patients presenting with clinical manifestations of CAD before age 50 often have predisposing risk factors. These risk factors are important for delaying/ identifying early onset of disease but less closely linked to the onset of coronary disease in later years

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It is now known that other abnormalities of lipids are also important in the pathogenesis of CAD, and these are looked for in patients with otherwise unexplained premature coronary atherosclerosis. Noteworthy amongst these are elevated levels of apolipoprotein(a) and of small, dense LDL lipoprotein particles. Evidence also suggests that hypertriglyceridemia is an independent risk factor for CAD. Elevated triglyceride levels often occur in association with other lipid abnormalities, including low levels of HDL cholesterol and elevated concentrations of lipoprotein(a).

One of the newer markers is Homocysteine. Elevated levels of serum homocysteine and non-specific markers of inflammation, such as cross-reactive protein (CRP), fibrinogen and ferritin, correlate with the occurrence of coronary disease. Hyperhomocysteinemia is known to increase the risk of thrombosis.

Pathophysiology:

Much is known today concerning the pathophysiology of atherosclerosis and the clinical presentations of CAD. Abnormal lipid metabolism or excessive intake of cholesterol and saturated fats – especially when superimposed on a genetic predisposition – initiates the atherosclerotic process. Low-density lipoproteins (LDLs) are the major atherogenic lipid. High-density lipoproteins (HDLs), in contrast, are protective and probably

assist in the mobilization of LDLs. The pathogenetic role of other lipids, including triglycerides, is less clear. LDLs undergo in-situ-oxidation, which makes them more difficult to mobilize as well as locally cytotoxic. LDL thus is more important marker of progression of CAD.

Primary & Secondary Prevention Of Ischemic Heart Disease

Although many risk factors for coronary artery disease are non-modifiable (gender, age, family history), active intervention in the modifiable ones like cessation of smoking, treatment of dyslipidemia and lowering of blood pressure can both prevent coronary disease and delay its progression and complications even after the disease is manifest. Treatment of lipid abnormalities delay the progression of atherosclerosis and in some cases produces regression. Even if there is no regression, fewer new lesions develop and coronary event rates are markedly reduced in patients with clinical evidence of atherosclerosis.

The importance of elevated LDL cholesterol in the pathogenesis of CAD – as well as stroke and peripheral arterial diseases – is substantiated by a series of trials. The substantial benefit of treating hyperlipidemia in preventing these conditions has also been proven beyond doubt. Trials have demonstrated improved outcomes even in patients who have already had myocardial infarction. The reductions were found in non-fatal cardiac events and cardiovascular deaths and the reductions occurred regardless of age, race or the presence of hypertension.

Aggressive lipid-lowering therapy should be implemented in all patients with Dyslipidemia and coronary artery or peripheral vascular disease. There is now clear evidence that reduction of LDL cholesterol can prevent coronary events & stroke in patients without clinically manifest atherosclerosis (primary prevention) and LDL levels as low as 130 mg/dL. A European study enrolled high risk patients with a mean LDL of 192 mg/dL and demonstrated a reduction of coronary death and nonfatal myocardial infarction.

Since oxidation of LDL appears to play a role in the atherogenicity of lipid molecules that have passed into the vessel wall, antioxidant therapy[1] has been advocated as a preventive measure.

Elevated plasma homocysteine levels are associated with an increased risk of vascular events. Although homocysteine levels can be reduced with dietary supplements of folic acid (1 mg/day) in combination with vitamin B6 and vitamin B12, it is not clear that this reduces clinical events in individuals with coronary artery disease.

Another preventive measure is aspirin prophylaxis.

The effect of hormone replacement therapy in postmenopausal women is uncertain. Epidemiologic data suggest that oestrogen protects against the development of coronary artery disease. However, the HERS trial, a prospective evaluation of estrogen use in women with coronary artery disease, showed no benefit in reducing mortality or preventing subsequent cardiac events. This issue should be considered unresolved.

Control of blood pressure has now been shown to prevent infarctions in older patients. Although unproved, it seems likely that control of blood pressure in younger individuals also prevents subsequent coronary events. The role of exercise remains controversial. Although individuals who exercise for at least 30 minutes a week are at lower risk for subsequent coronary events, it is difficult to be certain that this outcome relates specifically to exercise rather than a generally healthy lifestyle.

The decrease in number of coronary deaths over the last 2 decades may be due to a decrease in the prevalence of risk factors but probably also reflects improve-

ments in medical therapy, the role of coronary care units, better treatment of angina, arrhythmias and heart failure, and improved survival after coronary revascularization in some patient subsets.

Even at risk of insulting your intelligence, I would repeat the same for sake of completing the article.

1. Chest Pain in Coronary Artery Disease (*also discomfort, tightening, suffocation or uneasiness)*
Ischemic Pain more often then not is Sub-sternal *(not precordial)*. It is more like a pressure or tightening.
Ischemic Pain is NOT Pin Pointing, Pricking or Poking. It is MORE of Heaviness. Ischemic Pain may radiate to

- Jaw - Arm(s) - Shoulder(s)
- Tooth - Back

Any sub-sternal pain HAS to be thought of Coronary origin, unless proved otherwise. Intensity of the pain is NOT an important criterion to diagnose the cause of pain. There is NO co-relation between severity of IHD & severity of pain.
Very often a person with cervical spondylitis comes with retro sternal pain. How can we be sure of the diagnosis?
If the pain in the arm or at the elbow comes after a meal/ exertion/ emotional upset Coronary artery disease is to be suspected.
If pain comes on in the middle of the night, there is every reason to suspect angina. If pain comes on unaccustomed exertion and is relieved by rest, nitroglycerine or sorbitrate, Angina is the main suspect.
STABLE ANGINA - comes on accustomed exertion and is relieved by rest or a sublingual sorbitrate.
UNSTABLE ANGINA (aka Pre-Infarction Syndrome) comes on accustomed exercise or activity. In a known case of stable angina, the following may indicate worsening of stable to unstable angina
Change of Characteristic -
- Reduction of Effort Tolerance
- More intake of Nitroglycerine
- change in severity and duration of pain; change of referred point pain

MYOCARDIAL INFARCTION - Patient may present as -
- as a known case of angina not responding to Nitrates
- as a fresh case of IHD, 1st episode of chest pain

Pain in Myocardial Infarct is
- More intense pain than angina.
- Radiates to multiple referred points
- Accompanied by vomiting, Diarrhoea (due to autonomic disturbances), Bradycardia, Syncope, low BP

DIFFERENTIAL DIAGNOSIS OF INFARCTION
- Acute oesophagitis/ esophageal spasm
- Radicular Pain
- Hiatus Hernia
- Costochondritis
- FUNCTIONAL

UNUSUAL PRESENTATIONS OF ANGINA PAIN
- Pain on referred points
- Pain on [R] or posterior side (Inf or Lat wall Ischemia)
- Angina Equivalence symptoms
- *sweating (severe in Diabetics)*
- *sweating on exertion*
- *extreme weakness on exertion eg.*
- *breathlessness on walking*
- *extreme giddiness*

Diagnosis of Stable Angina
- History
- Resting ECG-[N] in 50% of patients in b/w episodes
- Cardiac Stress Test - +ve in 70%
- Holter - +ve only when pt gets chest pain

DIAGNOSIS
- Discovery of latent CAD
- Confirmation of clinically suspected CAD
- DD of Chest pain
- Evaluation of therapy
- Degree of LV dysfunction
- Judge Exercise capacity

Diagnosis of Status
-History
- Resting ECG
- Cardiac Stress Test / Thallium
- 2D-ECHO
- Coronary Angiography
- Holter monitoring
- Cardiac Enzymes

Cardiac Stress Test - Contraindications
- Suspected Unstable Angina
- Angina of recent onset
- BP = or > 220/110
- Cardiac failure
- Acute recent onset Mc Infarct
- Heart Block
- Systemic Infection

EVALUATION OF PATIENTS WITH ANGINA PECTORIS

A. Laboratory Findings: Serum lipid levels should be determined in all patients with suspected angina. The importance of LDL has already been reiterated.

NORMAL VALUES:

Cholesterol – Total - Normal < 200 mg %;
Borderline high 200-240 mg%; High > 240 mg%
Cholesterol – HDL - 35-60 mg %
Cholesterol – LDL - Desirable < 100 mg %;
Borderline 100-129 mg%; High risk > 130 mg%
Cholesterol – VLDL - 7-35 mg%
Chol/ HDL ratio - Low risk < 3.5; Moderate risk 3.5 – 5.0; High risk > 5
LDL/ HDL ratio - Normal range 2.5-3.5; High risk > 3.5
Triglycerides - 30-200 mg%

B. Electrocardiography: The resting ECG is normal in about 25% of patients with angina. In the remainder, abnormalities include old myocardial infarction, nonspecific ST-T changes, atrioventricular or intraventricular conduction defects and changes of left ventricular hypertrophy. During anginal episodes, the characteristic ECG change is horizontal or downsloping ST segment depression that reverses after the ischemia disappears. T wave flattening or inversion may also occur. Less frequently, ST segment elevation is observed; this finding suggests severe (transmural) ischemia and often occurs with coronary spasm.

C. Exercise Electrocardiography (Stress Testing): Exercise testing is the most useful noninvasive procedure for evaluating the patient with angina. Ischemic changes not present at rest are detected by precipitation of typical chest pain or ST segment depression (or rarely elevation). Exercise testing is done on a motorized treadmill. A variety of exercise protocol, are utilized, the most common being the Bruce protocol, which increases the treadmill speed and elevation every 3 minutes until limited by symptoms. At least two ECG leads are monitored continuously. In patients without baseline ST segment abnormalities or in whom anatomic localization is not necessary (where intervention is not planned), the exercise ECG should be the initial procedure because of considerations of cost and convenience.

a. Precautions and risks – The usually quoted risk of exercise testing is one infarction or death per 1000 tests. Patients who have angina at rest or minimal activity are at higher risk and should not be tested. Many of the earlier exclusions, such as recent myocardial infarction or congestive heart failure, are no longer valid if the patient is stable and ambulatory.

b. Indications – Stress testing is commonly used

a. to confirm the diagnosis of angina;
b. to determine the severity of limitation of activity due to angina;
c. to assess prognosis in patients known to have CAD, including those recovering from myocardial infarction;
d. to evaluate response to therapy; and
e. to screen asymptomatic populations for silent coronary disease. This indication is controversial as false-positive tests often exceed true positives, leading to anxiety and self-imposed disability or cardiac neurosis.

Exercise stress testing of asymptomatic patients should be done only for those at high risk (usually a strong family history of premature coronary disease or hyperlipidemia), those whose occupations place them or others at special risk (eg. airline pilots) and older individuals commencing strenuous activity eg a 40-45 yr old starting Gymnasium or playing Tennis/ squash.

c. **Interpretation** – The usual criterion for a positive test, 1 mm (0.1 mV) horizontal or downsloping ST segment depression (beyond baseline) generally picks up 60-80% of patients with anatomically significant coronary disease will have a positive test, but 10-30% of those without significant disease will also be positive. False-positives are uncommon with a 2-mm ST depression.

D. 2 D – ECHO: This is advised to pick up chamber enlargement, ventricular hypertrophy, wall motion abnormalities and abnormalities of cardiac valves.

1 250 gms daily of raw salads, vegetables and fruits.

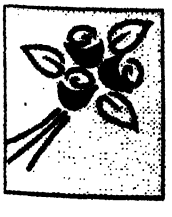
❑

The Heart Has Its Own Reasons

The Heart has its own reasons, that the human head knows not by Blaise Pascal. When the mathematical genius, the Jesuit priest called Blaise Pascal arrived at the above axiom, he presaged modern medicine's glorious ignorance about a malady commoner than greying of hair – loftily called Ischemic Heart Disease. As of today in the beginning of the 3rd year of the 3rd millennium, allopathy and all other pathies are unconscionably ignorant of the cause, course and cure of IHD.

To borrow Churchillian words, the human heart is a mystery wrapped by the puzzle inside an enigma. By the 4th week IUL, when the embryonic plate was just formed, the developing heart is the first to exhibit itself by an inbuilt throb. The cardiac tubes then create twins and turns, shunts and partitions, strings and valves and presto, create an indefatigable pump that will (ideally) beat 72 times a minute, 100,000 times a day and 4000,000,000 times in a lifetime of 100 years: Bravo, in the 100 years of top class unflagging service, it would work (contract) for 33 years, and literally relax (dilate) for 66 years. Even the cardiologists themselves have not learnt from the human heart how to relax and not chase the filthy lucre all the time.

Be as it may, all the animal torture in the name of heart research has not taken modern medicine beyond confused embryology/anatomy/physiology and carpentorial closure of septal defects and valve – replacement. About IHD medicine knows nothing and yet is predictably, able to confuse the bewildered common man.



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Cause of IHD:

Less said the better. There are now nearly 300 "predisposing" factors ranging from sex to sunshine, each such learned propositions arriving to botch up the gheeless meal with drops of saffola and scares the couple at night at time of androgenic flush. It's a jungle of proscriptions and atrocious prescriptions. The celebrated (but magnificently wasteful) Current Medical Diagnosis and Treatment (Lange, California), retouched and republished every year in yet costlier form, confesses that cholesterol–lowering "strangely" increases the mortality. Was it not Prof J S Bajaj of AIIMS who declared that IHD in India has shot up after Indians abjured cow-given ghee and took to man made cholesterol-lowering poisons?

COURSE OF IHD:

People with bad ECG survive the doctor. Normal coronary angiogram meets a sudden death, and "totally" blocked epicardial vessels allow the man to live into his eighties as revealed "incidentally" at a PM done for non-cardiac death. The revered pioneer cardiologist Rustom Jal Vakil, awarded Lasker prize for work on sarpagandha, at the fag end of his life, confessed that the ECG machine (and now of course the angiogram) has done far more harm to hassled mankind than even the atom bomb. The latest unliftable turns, twists the heart, now in its impressive 10th edition of 2001, admits that people granted clean cardiac chit, collapse and those doomed by medical prognosis join the condolence meeting of the cardiologists who had so predicted their death. A 400 page treatise on ICCU concluded that not one investigation reveals what the human heart will do, except saying that LUBB spells life, it's DUBB, the next LUBB is at the pleasure of God, or cosmos in case you are still an atheist.

CURE OF IHD:

All drugs, like in cancer, deprive coronaries of precious blood, by reducing the preload/stroke volume/ after load. Angioplasty/bypass rapes an innocent, virginal coronary field in a 1000 monstrous ways to pave way for shortened life, brain damage of sorts, and AIDS – Angioplasty Induced Defiant Stenosis. After 46 years of bypass and 36 of angioplasty, nobody has lived ½ hour longer as compared to the non-so-treated. The start is a travesty; for it lead-pipes every artery into which it is put, to occasion a new set of diseases called Malignant Instant Restonsis.

The best summing up of the current cardiologic scene is Sulman Rushdie's caustic comment in Satanic Verses, about a consultant visiting a rich man in Walkeshwar. The doctor, clad in a 3-piece suit in the sweltering heat of Mumbai, was "dripping with self-esteem". Have not the 6-figure fees of the cardiac field perverted the whole fee structure through and through?

By the way, what MIASMA will homoeopathy invoke, and what REPERTOIRE it will employ, and why? ❑

O Lord Hahnemann!

(Ed: A novice attempt in a local language- Hindi- by a young Homeopath is given here for your reading pleasure.)

O Hahnemann! You were the greatest physician of this world,
Cinchona se se tune Homoeopathy banaya, O Lord!!

Tune aisi pathy banayee
Allopath ko raas na ayee
Phir bhi tune zid na choda
Tune he khud ko Aude Sapere banaya!!
O Hahnemann.....(1)

291 Aphorisms ka
Tune Organon banaya!
Yeh Gita banake jag pe chaya
Tune hi Rapid Gentle Cure apnaya!!
O Hahnemann.....(2)

Psora, Syphilis, Sycosis ko
Fundamental cause bataya
Totality of symptoms ko apnakar
Tune suppression ka naam-nishaan mitaya!!
O Hahnemann.....(3) ❑

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