ISCHAEMIC HEART DISEASE

(Acute Myocardial Infarction)

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Introduction:-

Twenty-seven years back when I started my professional career in my place in India, the geographical conditions of which are very similar to Nepal, the cases of Myocardial Ischaemia there were practically unknown, but now it is found that the ischaemic heart disease in that place is as common as in other places of the country and the world. The geography of that place has not changed, but the social conditions and living habits of the people are all together different from what they were a quarter of a century back, probably due to the advance of the urbanisation which has now gripped that part of the country as much as it is making its sway into this lovely kingdom of Nepal. Since my arrival into this country about a year back, even though I have examined more than twenty-thousand cases uptodate, I have had no case of true Myocardial infarction so far except two, who were people of my country: but with the very rapid strides of advancement coming into this country bringing in fast changes in the living conditions and day to day life of the people here, and consequently increasing the stress and strain of life amongst them, one should anticipate that cases of myocardial infarction may not be so rare in about five to ten years from hence as they are now.

The basis of this paper is mostly derived from my observations of a series of 50 cases which came under my personal observation and treatment within a period of last eight years.

Definition:-

Acute myocardial infarction is a clinical syndrome resulting from sudden curtailment of the blood supply of the myocardium, and is

characterised by severe and prolonged cardiac pain and other symptoms and signs of cardiac damage and myocardial necrosis as evidenced by trelecocardiographis and laboratory changes.

Incidence:-

According to (1) Munck 80% of all sudden cardiac deaths are due to ischaemic Heart Disease. In the present day world it appears to be increasing rapidly all over.

Age & Sex:-

Cases have been recorded from 3rd decade of life to the eighth decade. Thus during the second World War (2) Newman and (3) Poe recorded cases of coronary artery disease in young soldiers between the ages of 22 to 26 years. According to (4) Cassidy 70% of the cases occur between 50 & 70 years of age, 14.6% between 40 & 50 years, 3.2% between 30 & 40 and 0.25% under thirty. In my series, 30 cases i.e. 60% were between the ages of 50 & 70; 15 cases i.e. 30% between 40 and 50 years of age and 5 cases i.e. 10% between 30 & 40 years of age. This is a great variance from the statistics of M. Cassidy who gave the above figures in 1946. Does it signify that the ischaemic heart disease is becoming in the lesser age group also now? One has to consider the problem very seriously. More men than women suffer from this disease. The general sex ratio given is 4:1 in favour of men but under the age of 50 it is 8:1. In my series out of the 30 cases between 50 & 70 years 26 were males and 4 females; while between 50 & 70 years, out of fifteen cases only one was female,

Habits & Occupation:-

The general impression that the people in the middle, upper middle and higher classes and especially professionals suffer more from ischaemic heart disease than the poorer classes, has much to appeal. The reason here may again be the stress of modern urban life. The other belief, that ischaemic Heart Disease is more a doctor's disease has also much to say. In my series except for a hosiery worker, I had none from the labour class. So far as doctors are concerned I had 11 cases of medical men under my care. Giving equal allowance to emotional tendencies and smoking etc. in peptic ulcer and coronary Heart Disease, peptic ulcer is consi-

common in taller thin persons, while myocardial infarction is believed to be attacking more people of short stocky build. Ischaemic heart disease these days seems to have disappeared in persons of size, stature and built, yet it will be interesting to keep it in mind especially in this country where the people are well-statured, well-built and stocky, and again in this country it is very prevalent in all sexes and in all ages and also in persons with factors like leucemic Disease, nutritional anaemia; hyperthyroidism and Ischaemic Heart Disease are not uncommon.

Dr. Sidney (4 & 5) in 1946 exonerated smoking but later (6) Doll & Hill in 1954 in England and (7) Hammond have shown that ischaemic Heart Disease is one and the same in common in cigarette-smokers than in non smokers.

Factors & Pathogenesis :-

Coronary Heart Disease signifies obstruction of the lumen of coronary artery by atheroma which reduces the cross-sectional area to less than one third of normal, and in some cases completely.

The most common cause is atherosclerosis of the coronary arteries, which progresses with age, but sometimes may be of moderate degree even in young adults. It is now believed to be closely related to disturbances of fat metabolism, acting over a long period of time. Advances in the Bio-Chemistry of blood lipoids; but it will be interesting to know the fatty substances in normal blood include neutral fat; cholesterol, cholesterol esters and phospholipids. The total serum cholesterol (which is normally 150 to 280 mg per 100 ml) is closely related to atherosclerosis but is believed to be only loosely related to blood lipid disturbance. According to (8) Gertler et al the phospholipid ratio was more closely related to atherosclerosis than the total serum cholesterol and this ratio is normally about 0.85 (phospholipid/cholesterol). This ratio is found to increase considerably in all conditions which encourage atherosclerosis e.g. Diabetes Mellitus, Myxoedema, Hypertension, High Blood Pressure etc. Consequently one finds that ischaemic heart disease is more common in diabetics and is said, that 40% of

Diabetics die of myocardial infarction. Ten of my fifty cases of all ages were diabetics.

Similarly aetrogens tend to restore the normal blood lipid pattern whereas androgens have the reverse effect (10) (Barr & Russ et al). Therefore bilateral oophorectomy in women increases atherosclerosis and also for this reason, by the age of 66-70 or so the Male/Female sex ratio in ischaemic heart disease is unity.

Site of Coronary Thrombosis & Infarction:-

The site and extent of the infarction depends upon three factors :-

- i) The B.V. or vessels occluded.
- (ii) Upon the capacity and efficiency of establishment of collateral circulation and.
- (iii) Upon the anatomy of the coronary circulation.

We all know that of the two main coronary arteries i.e.

(I) the left and (II) the right.

(I) The left coronary artery divides into:

- (a) a large anterior descending branch which runs down the interventricular groove to the apex of the heart and supplies (1) the anterior part of the Rt. Ventricle (2) the I.V. septum and (3) the anterior part of L.V.
- (b) The small left circumflex Branch which curls round the back between the Left Atrium and Ventricle and supplies the upper lateral and posterior basal portion of L.V.

(II) The right coronary artery does not divide but runs round to the back between the Rt. Atrium and Ventricle and supplies the:

- (i) the sinus node.
- (ii) Ant. part of R.V. and.
- (iii) Posterior base of both ventricles.

It will be seen, then, that the R.V. is supplied by the two big coronary arteries and is therefore the seat of infarction. Similarly the upper and lateral part of L.V. is supplied by the proximal

branches from both ant. descending and left circumflex arteries and is also relatively safe,

The posterior basal region is supplied by terminal branches only, some from the right coronary artery and some from the left circumflex, and is therefore less secure as compared to the R.V. and upper and lateral part of L.V., yet, it is still more fortunate than the anterior apex of L.V. which is supplied almost entirely by the terminal branches of the left coronary artery; though anastomosis can occur rapidly from the posterior descending branch of the Rt. coronary artery.

The I.V. septum is supplied anteriorly by the perforating branches of the ant. Descending artery and posteriorly by the perforating branches of the Rt. coronary artery.

Clinically we find that major coronary thrombosis involve the Anterior descending branch of the Left coronary artery in about 75% cases, the Rt. coronary artery in 25 to 40% and the left circumflex in 5 to 30% (110 Barnes & Ball, (12) Appelbaum & Nicolson).

It has been observed by (13) Wartman and Hellenstein in 160 cases that chiefly anterior infarction was seen in 72% and chiefly posterior infarction in 28%, but multiple in infarcts 41% cases. Half the anterior infarcts and a quarter of posterior infarcts, also, involved the I.V. septum.

Symptoms & Signs:-

Generally the onset of cardiac infarction is sudden. The outstanding feature is pain; very much similar to Angina pectoris but of much more prolonged type; lasting for hours. Again, like angina Pectoris it may or may not radiate. The pain is accompanied or soon followed by symptoms and signs of shock and collapse.

Clinically:-

The patient is in great agonising pain and is usually cold; clammy; sweating, extremely breathless; pale or cyanosed and collapsed or unconscious. Mild cases begin to look better within two or three days. During the attack, orthopnoea, cardiac asthma and pulmanary oedema have been observed. The B.P. falls rapidly to below 100 mm and is generally 80-90 mm on account of shock and remains such for several days: showing no

sign of recovery with treatment in cases who are to end fatally. The pulse is poor in volume and tension and often imperceptible. Cardiac sounds are faint. There may be paroxysmal ventricular tachycardia and signs of pulmonary oedema. Fever upto 101-102 F is commonly noted on the second day. There is leucocytosis with a T.I.C. count of generally 12000 to 15000 and high ESR, returning gradually to normal in a few weeks time.

Abnormalities in Serum Enzyme activity:

- (1) Serum Glutamic Oxalacetic Transaminase (S.G.O.T.) is a protein enzyme used in the synthesis of Glutamic acid and oxalacetic acid is normally present in high concentration in the myocardium, liver, skeletal muscle and brain. Its normal range of activity is 10-40 units. It is considerably increased in myocardial infarction in the first forty-eight hours; reaching a peak at about 24 hours or so and returning to normal between 2 to seven days. It is not increased in infectious, neoplastic, metabolic or degenerative diseases unless there is destruction of cardiac hepatic or muscular tissue. It is also not usually elevated in pulmonary embolism, peri-carditis or heart failure.
- (2) Lately (LDH) Lactic dehydrogenase activity in the serum is also being widely used especially in the United States and is believed to be of greater specificity than SGOT. LDH has been shown to consist of five components or isoenzymes 1,2,3,4,5, according to their electrophoretic speed of migration and are differentiated chromatographically. I use Pethidine 100 mg I.M. 4-6 hourly, because it is less constipating; causes lesser tendency to vomiting and has minimal effect on respiration. If the pain is persistent and so is sweating and restlessness or there is a falling B.P.; it indicates the need for *immediate* hospitalisation and treatment for cardiogenic shock.
 - (ii) For shock of mild nature bed rest, warmth and relief of pain as above.
 - (iii) For I.V. failure (cardiac asthma)
 - (a) morphine gr $\frac{1}{2}$ I.M. Inj.
 - (b) Digitalis in the form of digoxin 1 mg. I.V. in 10 cc of sterile water.
 - (c) aminophylline 500 mg. I.V. slowly over a period of five minutes.

I.V. Digoxin gives some relief within 20-30 minutes but the patient should not be left unattended unless asleep.

- (iv) Anticoagulant Therapy: start it immediately in all cases unless contra indicated e.g. in cases of peptic ulcer and liver disease and haemorrhagic diathesis. I usually give Heparin 20,000 units I. M. or in severe cases 10,000 units I.V. and 10,000 units I.M. and at the same time Dindevan 200 mg i.e 4 tablets of 50 mg each by mouth.

II. Treatment in Hospital.

It is essential for the treatment of the following conditions in acute myocardial infarction:-

- (1) Cardiogenic shock.
- (2) Thrombo embolic incidents.
- (3) Left Ventricular Failure.
- (4) Paroxysmal Ventricular Tachycardia and other serious arrhythmia

1. Cardiogenic Shock:

Severe shock is usually resistant to Therapy and, therefore, start treatment as soon as possibly by

- (a) Keeping the body comfortably warm by Blankets, but avoid excessive heating to avoid peripheral vasodilatation which lowers the B.P. further,
- (b) Giving oxygen @ 6-8 litres per minute by a simple plastic mask or through a nasal catheter or preferably an oxygen tent if available. It is valuable in combating anoxaemia which accompanies cardiogenic shock; and in cyanosis on account of pulmonary complication like acute pulmonary oedema.

It should be continued as long as the shock persists even though there may be no cyanosis.

- (c) There is no specific treatment for cardiogenic shock; but if the B.P. falls below 90 mm; vasopressor drugs should be used to raise it up so that the vital cerebral, renal and coronary circulations are maintained; and this cannot be done by increasing the circulating

blood volume with blood; Plasma, or saline transfusions because of the fear of overloading the diseased and wounded myocardium and on account of causing pulmonary oedema.

The vasopressor drugs enable the patient to survive the crisis till the myocardium recovers its strength to maintain the circulation.

- (i) Noradrenaline is the drug of choice. It raises both systolic and diastolic blood pressures without increasing the cardiac output in contrast to adrenaline which raises the blood pressure by its *direct* action on the myocardium increasing its rate and force of contraction. This is a great burden of hard work on the much too ill heart muscle and leads to dangerous cardiac arrhythmias.

Noradrenaline is given by drip in doses of 4-8 mg in 400-500 cc of isotonic saline or 5% glucose intra venously. 15-20 drops per minute are given and the B.P. is recorded every three minutes in the beginning and the drip is adjusted to maintain a B.P. of about 110/80 mm. Not more than 1500-2000 cc of fluid are given by drip, in 24 hours. While giving noradrenaline drip avoid the complications of Phlebitis and local tissue necrosis from leakage. Glucocorticoids like Decadron may be added to the noradrenaline drip if shock is not controlled.

Other vasopressors used are:

- (iii) Mephentin in doses of 15-30 mg diluted with 10 cc of distilled water may be given I.V. It can also be given intra-muscularly.
- (iii) Vasoxine dose 15 mg. and,
- (iv) Aramine dose 5-10 mg.
- (b) I.V. Digoxin or Ouabin in 0.5 mg dose I.V. in extreme emergency may be given. These drugs are especially indicated in pulmonary edema and congestive failure with cyanosis and dyspnea.
- (2) Thrombo-embolic incidents: are reduced by giving anticoagulants. Adjust their dose *daily* such that the Prothrombin time is maintained between 2 & 2½ times the control value, or prothrombin activity between 10% and 20% of the normal.

3 & 4 Arrhythmias:

The commonly encountered cardiac arrhythmias are:-

- (i) Ventricular tachycardia and Fibrillation.
- (ii) Premature beats.
- (iii) Paroxysmal atrial tachycardia, flutter and fibrillation.
- (iv) Heart Block.

- i) For Ventricular tachycardia and Fibrillation I.V. Digoxin is beneficial, which as already stated is useful in its prevention, and also in orthopnoea, pulmonary oedema (basal creps) and auricular fibrillation and flutter. If the pt. is already taking it; the drug should then be discontinued and quinidine 3 grs by mouth two hourly, or Procainamide 1 gm followed by 0.5 gm every 4 hours should be given. In critically ill patients Procainamide 1 gm dissolved in 10 cc of sterilized water can also be given I.V. very slowly i.e. @ 1 cc. per minute, and if possible under continuous electrocardiographic registration. In place of Procainamide, Quinidine Sulph 3 gr in 500 cc. of isotonic saline @ 50-100 cc per hou can also be given I.V.

Ventricular Fibrillation is usually a terminal event and in most of the patients hardly anything can be done by the time doctor arrives.

- ii & iii) For premature beats and Paroxysmal atrial tachycardia, flutter and fibrillation, again Quinidine Sulph or Procainamide are useful.
- iv) The various types of heart block that may occur from myocardial infarction rarely acquire treatment.

Rest

A word about fixed and strict bed rest may be said. It has been advocated for six weeks on the concept that this is the time required for the conversion of the infarct into a firm, healed scar, but this appears to be probably true only for the "transmural" infarct. My usual practice has been to recommend bed rest for only three weeks in uncomplicated cases and even during this time he is permitted to perform leg and foot exercises twice or thrice a day and feed himself as soon as the acute symptoms of the attack have subsided when he is also allowed to sit in bed or chair once or twice a day and if he

desires, he is allowed to use a bed side commode instead of bedpan. The bed rest and management during the next three weeks is gauged by (1) persistently high E.S.R. or W.B.C. count (2) Temperature (3) tachycardia (4) severe weakness (5) presence of complications like C.C.F., cardiac asthma, anginal pain etc. and (6) severe E.C.G. changes. Mental rest, is as important as physical rest and tranquilisers like Peritrate-meprobromate (Equanil) etc. should be given.

Long Term anticoagulant Therapy

I believe in giving anticoagulants for a long period of about 8 months to a year and possibly longer if needed, because (i) they lower the rate of further infarction (ii) decrease their severity (iii) and reduce the mortality (by about 50%). A large number of anticoagulants are available like Phenindione (also called 'Dividevan or Indema'), Tromexan, Dicoumrol, cyclocoumrol, Phenyl-propylhydroxycommarin etc. etc.

Out of all these Dividevan is preferable, for:

- a) It is more easily controlled.
- b) It is much less expensive.
- c) It is the long term anticoagulant of choice.
- d) Its effect on Prothrombin time is maximum in about 30 hours and it largely disappears in about 60 hours after a single dose. This duration of activity and lack of accumulative effect makes its administration easy to manage.
- e) it has few side effects, though G.I. disturbances and dermatitis may occasionally occur (and then one may switch on to Tromexan).

Doses:-

The maintenance dose of dividevan is 50-100 mg (1 to 2 tablets) in divided doses twice daily according to the prothrombin time estimation, because oral anticoagulants should usually never be given without regular estimation of Prothrombin time. Where on the spot facilities for Prothrombin time estimation are not available, clinical practice is to ask the patient to look for a dark urine and stop the drug as soon as it is noticed. As we all know, anticoagulants act indirectly by decreasing Prothrombin synthesis in liver. Stated above are my observations regarding the use of anticoagulant therapy in myocardial infarction. Many great cardiologists

have reported similarly e.g. (22) Wright, Marple and Beck in the Report of the Special Committee of the American Heart Association and (23) Gilchrist and Tulloch in U.K. It is also remarkable that many critical observers still feel uneasy about their reliability e.g. (24) William Evans in England and recently (25) Brown and (26) MacMillan in Canada.

Fibrolysis

These are Special Plasmin and strepto-kinase Preparations given by i.v. drip to hasten lysis of induced coronary thrombosis to reduce the size of the infarct and to promote healing, but their clinical use is still believed to be academic and experimental rather than practical.

Prevention of Ischaemic Heart Disease:

(The more civilised we become, the greater the risk of heart disease Dr. Radhakrishnan at the International Congress of Cardiology H.T. of 3.11.66)

The following factors may be considered in predicting the risk of Ischaemic Heart Disease:

1. Males above 40 years and women in post-menopausal period.
2. Family History of Hypertension, Ischaemic Heart Disease and Diabetes.
3. Presence of Diabetes, atherosclerosis or hypertension in the person.
4. Sedentary occupations.
5. Heavy smoking.
6. High Serum (B) lipoproteins and hypercholesterolaemia.

It is evident that neither age, nor sex nor family history of a person can be altered and therefore attention has to be focussed on other factors and these are :-

1. Avoid excess of smoking, drinking, eating and working.
2. Regular exercises within limits of tolerance
 - a) taking long walks daily should be made compulsory for those in sedentary occupations.
3. Correct obesity and overweight especially in Diabetes, hypertension,

etc. in those persons who have family of these diseases and of Ischaemic Heart Disease.

4. Diseases like hypertension and Diabetes should be properly treated and kept under control.

5. Mental relaxation is essential for those who are exposed to this risk.

6. Serum cholestrol should be kept in normal range and to do this:

(i) Reduce total fat content of the diet to less than 25% of the total calories.

(ii) Saturated fats like hydrogenated oil (Vanaspati) coconut oil, olive oil, animal fats like butter, ghee, should either be totally avoided or kept to the bare minimum, while unsaturated fats like groundnut oil, til, oil, safflower oil, corn oil (maize oil), pilchard oil, should form the bulk of dietary fats.

(iii) Administration of drugs like Nicotinic acid in high doses sitosterol, oestrogens, tocopherols (Vit E) and pyridoxine etc. have been advocated but they appear more of academic rather than practical interests.

In short a person running a risk of Ischaemic heart disease should have "moderation" as his watch-word in eating, smoking and drinking and should have also adequate physical exercise and mental rest.

Prognosis

21 out of my 50 cases are dead by now. 10 out of them died within 24 hours, six within three days and five within one week. Out of the ten cases who died within 24 hours five were diabetics, four hypertensive and one had no previous such illness. Again in this group three had attacks which were first and final; while seven patients died in their 2nd or 3rd attacks. The overall mortality, therefore, works out to be 42% and this agrees with the accepted rate of mortality. According to (16) Mintz and (17) Katz and Rusk and Zohman the following complications adversely influence the mortality:-

Complete heart block (80-90%), shock (75-80%), Paroxysmal tachycardia of any type or atrial flutter (66%) Pjeumonia (57%) L. Ventricular r C.C.F. (50%) gallop rythm, a pulse pressure below 20 mm Hg. a B.P.

under 90 mm. intractable pain, diabetes mellitus and obesity. Regarding Diabetes Melitis it will be interesting to note that recently H. H. Marks of the Joslin Clinic, Boston, who studied the longevity, mortality and causes of death of some 50,000 (fifty thousand) diabetics has reported (in the Amer. J. Publi. Hlth. Vol 55, March 1965) that cardi-vascular Disease, which was the certified cause of death in less than 50% of cases in 1922 now accounts for three-quarters of diabetic deaths, heart disease alone accounting for more than half.

Electrocardiographically favourable cases are those which show only T wave inversion, without pathological Q waves and without initial elevation of R. S. T. segment ((18) East & Oram) and when there is no significant fever; leucocytosis and raised E. S. R. (. (19) Helander).

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