ANGINA PECTORIS WITH NORMAL CORONARY ARTERIES

By Lim Chin Hock

SYNOPSIS

A 23 year old Chinese female with angina pectoris and normal coronary arteries is described. She has a changing electrocardiographic pattern. The pathogenesis, natural history and prognosis is briefly discussed.

Selective coronary arteriography (Sones, 1962), recently introduced in Singapore, is a useful and well-established procedure for the diagnosis, evaluation and management of patients with ischaemic heart disease. Its introduction has dispelled the common belief that angina pectoris and myocardial infarction is invariably the result of coronary artery disease. Angina pectoris can occur in patients with severe valvular stenotic lesions (e.g. aortic stenosis, mitral stenosis, pulmonary stenosis), in hypertrophic cardiomyopathy, severe aortic incompetence, syphilitic aortitis or severe anaemia, and it occurs in some 10% of these patients presenting with chest pain superficially suggesting angina pectoris (Kemp, 1967; Likoff, 1967; Ross, 1966). Acute myocardial infarction too can occur in individuals with normal coronary arteries (Likoff, 1962; Ross, 1966; Sidd, 1920; Glaney, 1971; Bruschke, 1971; Khan, 1974). Conversely, complete coronary artery occlusion can occur, and yet does not produce symptoms of myocardial infarction (Blumgart, 1940). There is no single satisfactory explanation why angina pectoris or myocardial infarction can occur in individuals with normal coronary arteries (Editorial, Lancet, 1974; Editorial, New Eng. J. Med., 1974). There are no characteristic features which may suggest its presence (Kemp, 1973). The purpose of this paper is to report a case of angina pectoris with normal coronary arteries occurring in a 23 year old Chinese female and to draw attention to this condition which may not be uncommon in our population if coronary arteriography is done more frequently.

CLINICAL SUMMARY

A 23 year old Chinese woman was first seen on 8.5.74 for palpitations, vomiting and giddy spells of 2 months' duration. She was noticed to be pale and was thought to be on the verge of death during these episodes. These episodes were accompanied by retrosternal chest discomfort which occurred both at rest and on exertion. She did not smoke or take oral contraceptives. There was no past or family history of importance. The only positive finding was the presence of arcus juvenilis. B.P. was 100/60.

Laboratory investigations were all normal. Haemoglobin, haematocrit, serum lipids, serum enzymes, urine microscopy, blood urea, blood sugar and uric acid levels were within the normal limits.

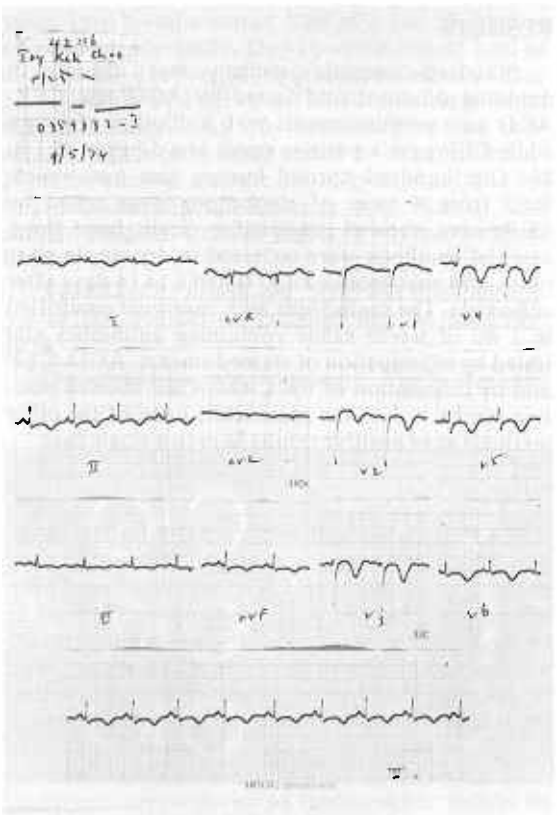

Fig. 1. 12-lead ECG done in May 1974 showing global ischaemia and simulating anteroseptal infarction. There is widespread ischaemic T wave inversion and ST segment depression.

Department of Medicine, University of Singapore.

LIM CHIN HOCK, A.M., M.B., B.S., M.Med., F.R.A.C.P., Senior Lecturer.

Correspondence: Dr. Lim Chin Hock, Department of Medicine (II), University of Singapore, Sepoy Lines, Singapore 3.

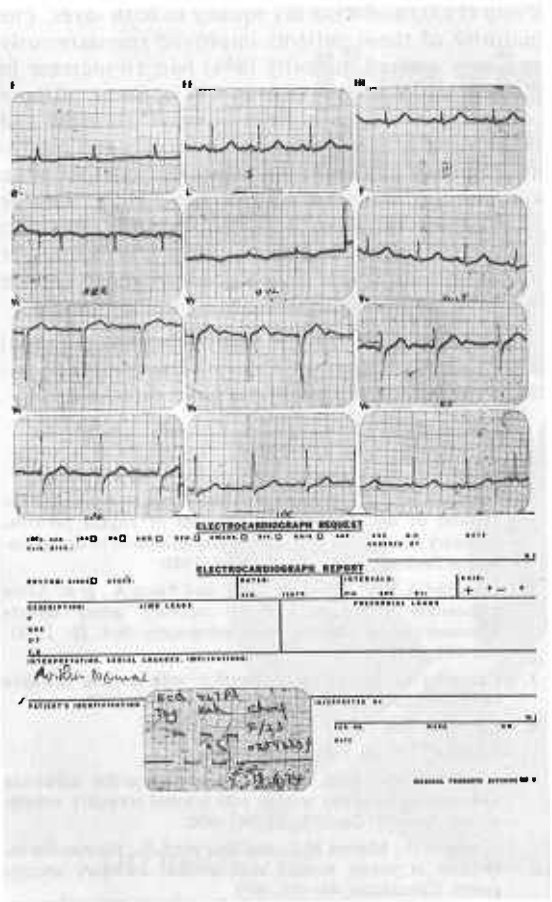

Fig. 2. 12-lead ECG done in June 1974. Apart from nonspecific T wave inversion in III, this ECG is within normal limits.

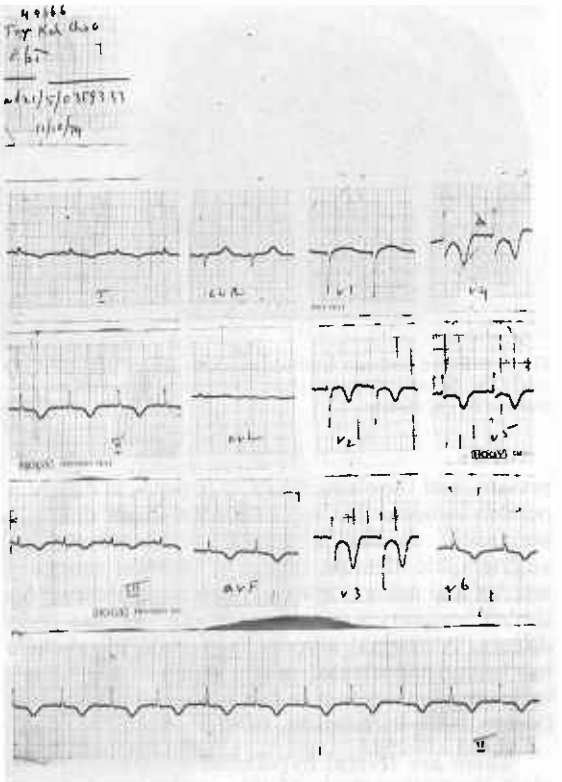

Fig. 3. 12-lead ECG done in October 1974 shows gross ischaemic changes with prominent T wave inversion in the praecordial lead.

LE cell. were negative and the chest X-ray was normal.

Serial ECG's done from May 1974 showed a changing ECG pattern. It was grossly abnormal in May 1974 (Fig. 1) showing a pattern of global ischaemia with T wave inversion in leads I, II, III, aVF, and V2 to V6. ST segment was depressed in II, III, aVF, V4 and V5, and there was poor R wave progression from VI to V6. The ECG reverted to normal from June to August 1974 (Fig. 2). In October 1974, it again showed ischaemic changes similar to that seen in May 1974 (Fig. 3). In November 1974, these changes became less pronounced and there were only flat T waves in I, aVL and from V4 to V6.

Right heart catheterisation and selective coronary arteriography using the Judkins percutaneous transfemoral method was done on 22.7.75. The right and left heart pressures were normal and the cardiac index was 3.2 L/min/m^2. The right and left coronary arteries and its branches were completely normal (Figs. 4 and 5). The left ventricular angiogram revealed normal left ventricular contractility.

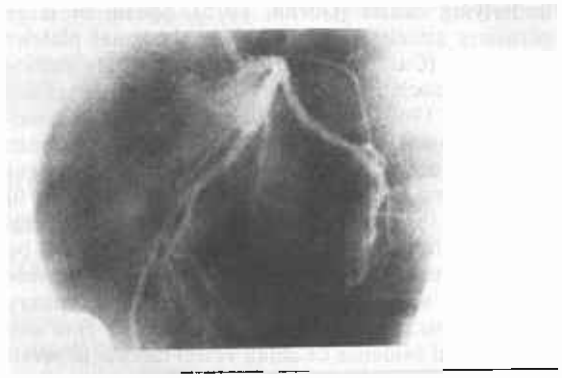

Fig. 4. Selective arteriography of the left coronary artery shows normal main left coronary artery, the left anterior descending, diagonal branch and circumflex arteries.

DISCUSSION

Although this patient did not have the classical Heberden type of angina i.e. severe and crushing retrosternal chest pain on effort and associated with angor animi, the episodes of retrosternal chest discomfort, associated with facial pallor and electrocardiographic evidence of global ischaemia indicates that she does indeed have angina pectoris from myocardial ischaemia. There are no coronary risk factors

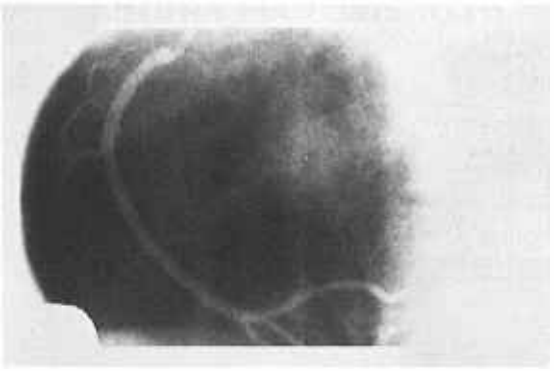

Fig. 5. Selective coronary arteriogram shows a large right coronary artery free of luminal irregularities. Its major branches appear normal and free of disease.

present, and the changing ECG patterns at different periods indicate that the ischaemia is not due to a permanent cardiac abnormality. Moreover, the angiographic demonstration of normal coronary arteries and normal left ventricular morphology excluded coronary artery disease and valvular heart disease. Abnormal myocardial lactate metabolism has been demonstrated in this group of individuals indicating that there is in fact myocardial ischaemia (Kemp, 1973; Richardson, 1974).

There are several hypotheses attempting to explain the pathogenesis of this syndrome. A forme fruste variety of idiopathic hypertrophic subaortic stenosis, and the prolapsing mitral valve syndrome have both been postulated to be the most likely underlying causes (Gorlin, 1975). Spasm of large coronary arteries (Lange, 1972), abnormal platelet behaviour (Campeau, 1970), abnormal oxyhaemoglobin dissociation and oral contraceptives (Eliot and Bratt, 1969) have also been advocated as possible aetiological causes. Abnormal oxyhaemoglobin dissociation or the stingy-Hb concept is at present not accepted by most workers. James (1967, 1970) postulated that angina in these cases could have been due either to small vessel disease which could not be demonstrated angiographically with our present techniques or to misinterpretation of the coronary arteriograms. Richardson (1974) could not find any histological evidence of small vessel disease in seven patients with angina pectoris and normal coronary arteries. The only abnormal histological finding was attenuation of hypertrophied myocardial fibres.

The clinical syndrome of angina pectoris with normal coronary arteries has been well described by Kemp (1973) and it occurs equally in both sexes. The majority of these patients improved spontaneously and only a small minority (8%) had an increase in the severity of angina (Kemp, 1973). Some of these patients derive benefit from glyceryl trinitrate and beta blockers. It has a benign prognosis and mortality from this condition is not greater than that of an age and sex matched control population. Deaths have, however, been reported in this condition (Kemp, 1973, Eliot and Bratt, 1969). It is likely, that this patient too would have a good prognosis despite the alarming resting electrocardiographic changes.

This case illustrates the great value and potential of coronary arteriography in the management of patients with suspected ischaemic heart disease.

REFERENCES

1. Blumgart H.L., Schlesinger M.J. and Davis D: Studies on the relation of the clinical manifestations of angina pectoris, coronary thrombosis and myocardial infarction to the pathological findings. Amer. Ht. J., 19: 1, 1940.
2. Bruschke A.V.G., Bruyneel K.J.J. and Bloch A., *et al:* Acute myocardial infarction without coronary artery disease demonstrated by selective cinearteriography. Brit. Ht. J., 33: 585-594, 1971.
3. Campeau L: Myocardial infarction with normal selective coronary arteriograms. Amer. Ht. J., 79: 139, 1970.
4. Editorial: New Eng. J. Med., 291: 470, 1974.
5. Editorial: Lancet, vol. II, 702, 1974.
6. Eliott R.S. and Bratt G: Paradox of myocardial ischaemia and necrosis in young women with normal coronary arteriograms. Amer. J. Cardiol., 21: 98, 1968.
7. Glaney L.D., Marcus M.L. and Epistein S.E.: Myocardial infarction in young woman with normal coronary arteriograms. Circulation, 44: 495, 1971.
8. Gorlin R: Personal communication, 1975.
9. James T.N.: Pathology of small coronary arteries. Amer. J. Cardiol., 20: 679, 1967.
10. James T.N.: Angina without coronary disease. Circulation, 42: 189, 1970.
11. Kemp H.G., Vokonas P.S., Cohn P.F. and Gorlin R.: The anginal syndrome associated with normal coronary arteriograms. Report of a six year experience. Amer. J. Med., 54: 735, 1973.
12. Khan A.H. and Haywood L.J.: Myocardial infarction in patients with patient coronary arteries. New Eng. J. Med., 291: 427, 1974.
13. Likoff W., Segal B.L. and Kaspareau H: Paradox of normal selective coronary arteriograms in patients considered to have unmistakably coronary heart disease. New Eng. J. Med., 276: 1063, 1967.
14. Richardson P.J., Lively B. and Oram S., *et al:* Angina pectoris with normal coronary arteries. Lancet, II: 677, 1974.
15. Ross R.S. and Friesinger G.C.: Coronary arteriography. Amer. Ht. J., 72: 437, 1966.
16. Sones F.M. and Shirey E. K.: Cine coronary arteriographs. Mod. Concepts Cardiovasc. Dis., 31: 735, 1962.