# Anterior And Inferior Myocardial Infarction With Normal Coronary Arteries

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## Summary

**Myocardial infarction with angiographically normal coronary arteries is being increasingly encountered in younger patients. Such cases have an excellent long term prognosis and are to be managed conservatively. We present one such case that we came across.**

Myocardial infarction is commonly considered to be the end result of atherosclerotic heart disease where atheromatous plaque rupture and intraluminal thrombus formation is the likely pathophysiological mechanism[1]. Occasional reports of myocardial infarction with 'Normal Coronary Arteries' are available in Western literature[2,3]. We are reporting one case of myocardial infarction with normal coronary arteries from the Indian subcontinent.

## Case Report

A 26-year old man presented with history of sudden severe retrosternal chest pain at rest of more than half an hour's duration associated with profuse sweating 6 months ago. He was normotensive, nondiabetic and a chronic smoker. He had no family history of Ischaemic heart disease. His serial E.C.Gs confirmed the diagnosis of inferior and anterior myocardial infarction (Fig. 1) alongwith raised cardiac enzymes. The patient was managed conservatively at a peripheral centre and was not given any thrombolytic agents. He was referred to us for further investigations.

Clinical examination at the time of admission revealed a regular pulse rate of 80 per min. Other peripheral pulses were similarly palpable. His blood pressure was 122/80 mm. Hg and respiratory rate was 18 per minute. Jugular venous pressure was not raised. Examination of precordium revealed an apex beat in 5th intercostal space in mid clavicular line which was diffusely felt with double contour. Both heart sounds were normally present with audible $S_4$, without any murmur or rub. Chest was bilaterally clear. There was no hepatosplenomegly or any free fluid in abdomen. His ECG revealed evidence of healed inferior and anterior myocardial infarction with pathological Q waves in inferior and septal leads (Fig. 1). His hemogram revealed Haemoglobin of 15 gm.%, packed cell volume of 46%, Bleeding time was 4 minutes 30 seconds and platelet count was 4.2

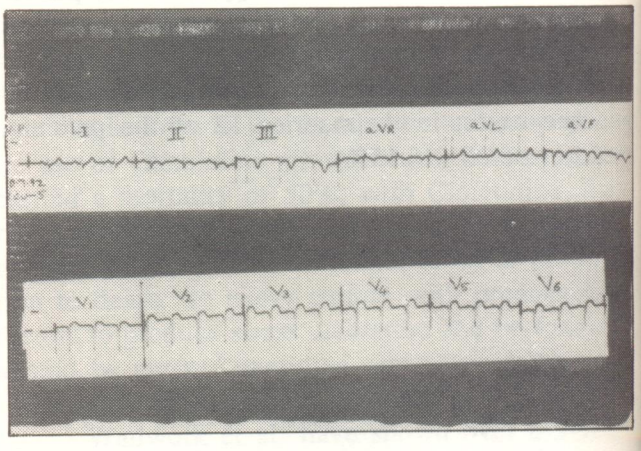


Fig. 1
Electrocardiogram showing healed inferior and anterior myocardial infarction.

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lacs, clotting time 7 minutes 2 seconds, partial thromboplastin time was 48/45 seconds. His biochemistry revealed fasting blood sugar of 86 mg.%, post prandial blood sugar of 136 mg%, serum urea

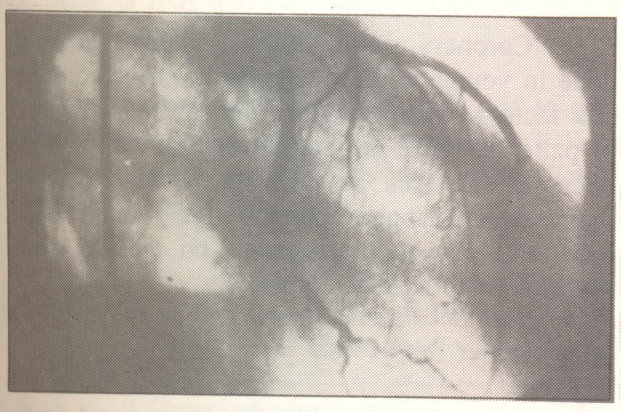

Fig. 2
Left coronary angiography showing normal origin of left coronary artery (Type III) and no atherosclerotic narrowing.

38 mg%, total serum cholesterol 216 mg%, high density lipoprotein 56 mg%, low density lipoprotein 159 mg% and serum triglyceride 214 mg% and serum fibrinogen level was 300 mg%. On treadmill stress testing (Bruce Protocol), the patient exercised for 6 minutes, 32 seconds and attained 70% of maximal predicted heart rate at 7METS. Flat ST depression of 0.8mm. in lead aVL was noted at peak exercise. 2D Echocardiography showed dyskinesia of left ventricular apex, mild hypokinesia of posterior wall with an ejection fraction of 54%. Cardiac valves were normal. A 24 hours Holter recording did not show any episode of symptomatic or asymptomatic significant ST depression. Cardiac catheterization revealed a LVEDP of 18 mm Hg. There was no gradient across the aortic valve. Left ventricular angiogram showed antero-lateral, apical and distal inferior wall akinesia with an ejection fraction of 48%. Coronary angiography showed normal origin of left and right coronary arteries from their respective sinuses without any atherosclerotic plaque. Left anterior descending artery was a large vessel which curved round the apex and continued on the inferior surface (Fig. 2 & Fig. 3).

## Discussion

Ischaemic heart disease has been considered to be the consequence of atherosclerotic obstruction in the coronary arteries, which renders them unable to meet the increased metabolic demands of the heart[1]. Recently it has been observed that in about 6% of patients of acute myocardial infarction, coronary arteriography reveals normal coronary arteries[4]. This small subset of patients usually belong to a younger age group. Perhaps 20% of patients under the age of 35 years do not have coronary atherosclerosis demonstrated by angiography on autopsy[1]. These patients have relatively fewer coronary risk factors; though most are chronic smokers[5,6,7]. Usually they have no history of angina prior to myocardial infarction[8].

In a case of acute myocardial infarction with normal coronary arteries various explanations are worth considering. First, is a false diagnosis of acute myocardial infarction[3]. Diagnosis of myocardial infarction in the case that we report cannot be doubted in view of the classical chest pain, characteristic serial ECG changes and serial cardiac enzymes during the acute episode alongwith residual changes of old infarct. Hypertrophied hearts with left ventricular outflow obstruction and hypertrophic cardiomyopathy can also develop acute myocardial infarction with normal coronary arteries[3]. Such factors were not present in our case. A variety of haematological disorders may cause in situ thrombosis in the presence of normal coronary arteries such as polycythaemia, sickle cell anaemia, disseminated intravascular coagulation and thrombocytosis[9]. Infarction could not be attributed to any such cause in our case.

The coronary arteriograms could be misin-

Fig. 3
Right coronary angiography showing normal right coronary ostium and disease free right coronary artery.

terpreted[3,6,7]. It is a well established fact that coronary angiograms tend to underestimate the degree of coronary arterial narrowing. Technical factors such as equipment, clarity of films, number of views as well as the experience of the angiographer are important in the accurate interpretation of angiographic films. Post-mortem studies have shown that significant coronary artery disease may be present and missed by competent coronary angiographers. The two commonest causes of this are slit like crescentric lumen caused by eccentric plaques and occlusion of an artery at its origin from the parent vessel. In the above mentioned case, the angiogram was reviewed independently by two experienced invasive cardiologists. Patients with anomalous origin of coronary arteries, coronary arteriovenous fistula and myocardial bridges may also sustain myocardial infarction in the absence of atherosclerotic narrowing. No such causes were found in our case.

Acute myocardial infarction may be caused by an occluding embolus which may lyse spontaneously producing a recanalized vessel[3,4,6,7]. Histological studies of previous known arterial emboli indicate that such lesions may either lyse completely or retract or recanalize to produce a lumen large enough to be viewed as a normal coronary arteriogram[3]. Although its source may not be known, coronary embolism may be more common than is generally assumed.

Coronary spasm may also cause myocardial infarction. Several investigators have identified the importance of coronary vasospasm as a cause of angina and myocardial infarction[6,7]. Just as normal coronary arteries do not rule out the possibility of myocardial ischaemia caused by coronary vasospasm, the presence of organic stenosis does not necessarily prove the organic pathogenesis of myocardial ischaemia. We did not attempt a provocative test with ergonovine in our case. This test is not free from complications in a setting of prior myocardial infarction and is not positive in all cases of spasm[6]. Our case did not have prior angina and holter recording also did not show any symptomatic or asymptomatic ST elevation, which are frequent in patients with repeated coronary spasms. Smoking has been shown to precipitate coronary spasm[10] and it may have caused vasospasm in our patient. Intravascular thrombosis is also commoner in smokers. A combination of these two factors might have led to occlusive coronary event and the lesion may have completely lysed by the time of angiography.

Coronary angiography should always be done in young patients who do not have coronary risk factors. It may help in identifying a subgroup of patients where normal coronary arteries can be documented. This assumes great importance as they can be managed conservatively and have an excellent long term prognosis on medical treatment[5,6].

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