Coronary artery disease in a young man with normal serum lipids

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A young patient is described in whom cardiac infarction occurred despite the virtual absence of the risk factors usually associated with this condition.

Coronary heart disease tends mainly to present after the age of 40, but the incidence may be rising in younger subjects (Friedberg, 1966). So few cases occur under 30 that one should seek possible underlying metabolic or anatomical causes, which are especially evident in young patients (Heinle *et al.*, 1969; Tzagournis *et al.*, 1968).

Case report

The patient was born in Israel of Yemenite Jewish parents, and has lived in England since 1962. On 30 August 1968, when 23 years old, he experienced squeezing retrosternal pain for 4 hours, and was admitted to hospital. None of his family has suffered from ischaemic heart disease. He smoked 10 to 15 cigarettes a day and followed a conventional Western diet. He worked as a motor mechanic; there was no history of trauma to the chest.

He was 169 cm tall and weighed 63.5 kg. There were no physical abnormalities on examination; the blood pressure was 120/70 mmHg, with no evidence of pre-existing hypertension. The peripheral pulses were normally palpable. Electrocardiograms showed the evolution of partial-thickness anterolateral cardiac infarction: on the day after admission there were inverted T waves in leads I, aVL, V5, and V6, and ST depression in V4 (Fig. 1). Urinalysis, full blood count (Hb 16.3 g/100 ml), erythrocyte sedimentation rate, blood urea, electrolytes and proteins, and chest x-ray were all normal. The serum enzymes are shown in Table 1.

Treatment included analgesics, rest, intravenous heparin and oral warfarin, and he stopped smoking. Over the next two weeks the abnormalities on the electrocardiogram improved, though T inversion in leads I and aVL was still present after 8 weeks. He recovered rapidly and

FIG. 1 Electrocardiogram at onset of illness, 31 August, 1968.

was discharged from hospital one month after admission. During the next 6 weeks he had several mild episodes of angina on exertion; since then he has been symptom free, and has remained normotensive (blood pressure range 105-125/70-80 mmHg). The electrocardiogram had returned to normal after 4½ months, but there was

TABLE 1 Serum enzyme values

	SGOT (Frankel units)	SGPT	LDH International units
30 August	46	38	420
31 August	100	35	675

plane ST depression in postexercise tracings recorded one and two years later.

Further investigations have shown low-normal serum lipids (Table 2); on thin-layer chromatography of plasma lipids, the ratio of ester to free cholesterol appeared within normal limits. Glucose tolerance and plasma insulin response were normal after 50 g glucose orally (Table 3). The serum uric acid was 3.3 mg/100 ml: Wassermann reaction, lupus erythematosus cells, and antinuclear factor negative. On 14 April 1970, cardiac catheterization was normal; coronary angiography showed: left coronary artery—proximal narrowing of anterior descending and circumflex branches (Fig. 2); right artery normal, contributing collaterals to the ischaemic area.

Discussion

The patient presented with the clinical features of cardiac infarction; this was confirmed by the electrocardiogram and serum enzyme changes. Subsequently further evidence of cardiac ischaemia was obtained from the positive effort cardiograms, and occlusive coronary artery disease was demonstrated by angiography.

The importance of abnormalities of lipoprotein and carbohydrate metabolism in younger subjects with coronary artery disease has been stressed by Heinle *et al.* (1969): 80 per cent of their cases aged less than 50 had hyperlipoproteinaemia (including all aged 20–29); 96 per cent had one or other abnormality. Our patient, who developed signs of ischaemic heart disease at the age of 23, is strikingly unusual: there is no hyperlipidaemia and his glucose tolerance is normal. Furthermore, he is not obese (body weight within the normal range of the Metropolitan Life Insurance Co., 1959), has always been physically active, has consistently had a normal blood pressure, the serum uric acid is normal, and there is no family history of premature vascular disease; nor were there indications of psychological stress. Cigarette smoking was the only specific risk factor present (Stamler, 1971).

Not only did he not have hyperlipoproteinaemia: his serum lipid levels tended to be low. Furthermore, he belongs to an ethnic group in which coronary artery disease is relatively uncommon, whether for genetic or environmental reasons (Krikler, 1970; Vlodaver, Kahn, and Neufeld, 1969). Despite this, he suffered a cardiac infarct at an extremely young age, showing no evidence of inflammatory arteritis, and having no signs of aortic valve disease or of an abnormal origin to either of his coronary arteries. Though histological proof of the diagnosis of coronary

FIG. 2 Left coronary arteriogram, left anterior oblique projection, showing (a) narrowing of anterior descending branch, suggesting recanalized thrombosis, and (b) stenosis of circumflex branch, with poor filling distally.

TABLE 2 Plasma lipid values

	Cholesterol (mg/100 ml)	Triglycerides (mg/100 ml)	Lipoprotein electrophoresis
30 August 1968	125		
16 September, 1968	170		
7 February, 1969	140	62	
17 December 1969	100	74	Low β , no pre- β } α and β visible
10 July 1970	160	49	
17 July 1970	135	56	

Preparative ultracentrifugation:

	Cholesterol (mg/100 ml)	Triglycerides (mg/100 ml)
Very low density lipoproteins (D = 1.006)	10	18
Low density (β) lipoproteins (D = 1.006–1.063)	114	21
High density (α) lipoproteins (D = 1.063–1.21)	49	20

TABLE 3 Glucose tolerance test and serum insulin values

	Blood glucose (mg/100 ml)	Serum insulin (μU/ml)
Fasting	77	6
30 minutes	126	34
60 minutes	110	7
90 minutes	100	—
120 minutes	54	—

atherosclerosis is lacking, the features are otherwise quite in keeping with this diagnosis. The exceptional feature of our case is that most of the known risk factors for ischaemic heart disease were absent in a patient presenting with myocardial infarction at the age of 23.

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