HYPERTENSIVE HEART DISEASE OR ACUTE CORONARY SYNDROME?

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CASE REPORT

A 66-year-old Afro-Caribbean woman, who had a known history of long-standing hypertension and was being treated with nifedipine retard 20 mg b.i.d., was admitted with a history of chest pain. The latter lasted 30 minutes and settled with sublingual nitrates administered in the Accident and Emergency Department. There was no previous past history or family history of ischaemic heart disease. There was no evidence of connective tissue disease or diabetes. She was a non-smoker and her serum cholesterol was 4.2 mmol/1. She was started on subcutaneous enoxaparin with presumed diagnosis of unstable angina.

On examination she was overweight and her blood pressure on admission was 191/88mmHG. Her initial ECG on admission showed lateral T-wave flattening (Figure 1), but by the following day showed anterolateral T-wave inversion (Figure 2). There was no evidence of LVH by voltage criteria. Despite her ECG changes, her serial cardiac enzymes (creatinine kinase and aspartate alanine transferase) (Table 1) remained within the normal range. She remained pain free following her admission, but due to a recent total hip replacement, the patient was unable to perform a treadmill exercise tolerance test.

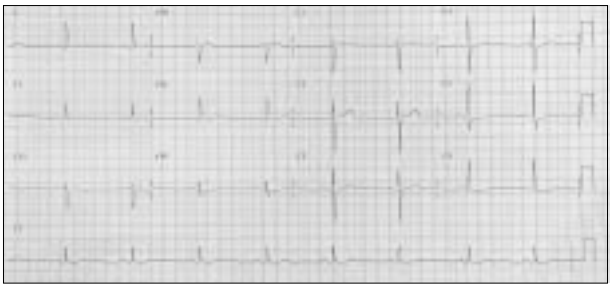

FIGURE 1
12-lead ECG on admission, showing ST and T wave flattening laterally.

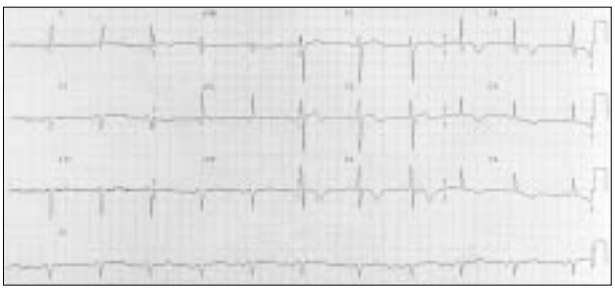

FIGURE 2
12-lead ECG on Day 2, showing marked T wave inversion anterolaterally.

Cardiac catherterisation and coronary angiography were therefore performed, which demonstrated normal coronary arteries (Figure 3) and normal left ventricular function, with no evidence of valvular heart disease of cardiomyopathy. The left ventricular end-diastolic pressure was 18mmHg. There was no evidence of coronary artery spasm. In order to rule out the possibility of hypertrophic obstructive cardiomyopathy (HOCM) or other structural heart disease, a transthoracic echocardiogram and cardiac Doppler was also carried out. This confirmed concentric left ventricular hypertrophy (LVH) without any evidence of HOCM or valvular heart disease (Figure 4). The patient was discharged home on Tildiem (diltiazem) LA300mg 1 tab o.d and at follow-up three months later, she remained well with a blood pressure of 130/70mmHg.

TABLE 1
Sequential cardiac enzyme changes

	Day 1	Day 2	Day 3
Creatine kinase IU/1 (normal range <150)	107	80	76
Aspartate transaminase IU/1 (normal range <60)	24	20	24

DISCUSSION

Black hypertensives have, compared to whites, a higher prevalence of LVH, with its associated higher risk of cardiovascular morbidity and mortality.[1-3] LVH is also known to interfere with diagnostic accuracy of the ECG exercise tolerance testing in suspected coronary artery disease, making differentiation between 'strain' pattern and ischaemic changes sometimes unachievable. As the present case illustrates, the ECG may also mimic an acute coronary syndrome. However, we cannot exclude underlying coronary artery thromboembolism as the cause of her initial presentation. Indeed, myocardial infarction with normal coronary arteries at coronary angiography can occasionally occur,[4] but in the present case, our patient's cardiac enzymes did not suggest any myocardial damage and thus the ECG changes are unlikely to be due to a non-Q myocardial infarction. The patient was also taking a calcium antagonist at presentation, which tends to ameliorate coronary artery spasm, if this was the underlying diagnosis. Furthermore, she has had few symptoms at follow-up following good blood pressure control.

Despite the increased risk for ischaemic heart disease in hypertensives with LVH, normal coronary angiography findings are not a rare discovery. This discrepancy between ischaemic changes on ECG and macroscopically normal coronaries has several underlying pathophysiological mechanisms. These include structural changes of the

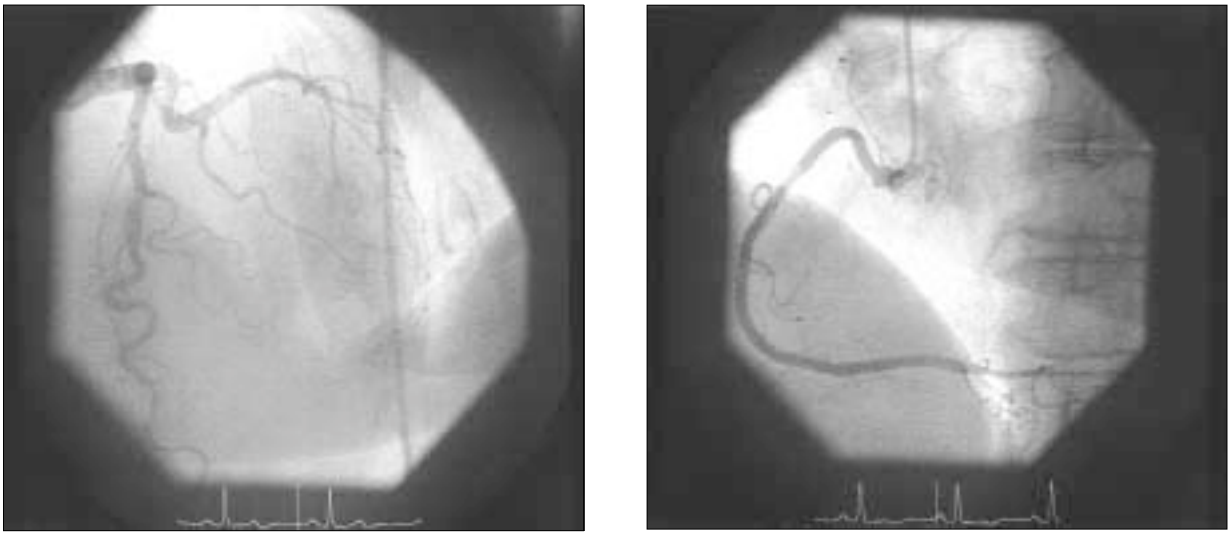

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FIGURE 3
Coronary angiography showing normal (a) left and (b) right coronary arteries.

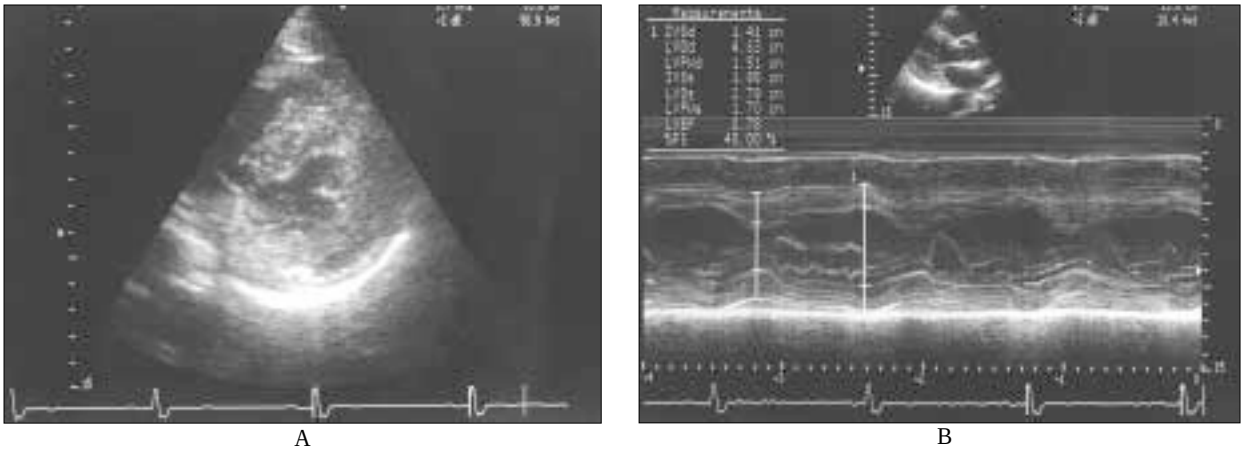

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FIGURE 4
(a) Two-dimensional and (b) M-mode echocardiography showing concentric left ventricular hypertrophy.

microvasculature,[5] endothelial dysfunction,[6] as well as a relative reduction in the capillary network in LVH.[7] Indeed, there is substantial evidence of reduced coronary flow reserve in LVH, even in the absence of macroangiopathy. These changes are at least partially reversible after normalisation of the pressure overload by good blood pressure control, [8, 9] thus emphasising the importance of good blood pressure control in hypertensives, even without angiographic evidence of coronary artery disease.

References

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