Direct measurement of low-density lipoprotein in diabetic patients with end-stage renal failure

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Calculation of serum low-density lipoprotein (LDL) from the Friedewald equation is used commonly in UK laboratories. However, a fasting triglyceride sample is required for the Friedewald equation to apply, which can be difficult in diabetic patients. Moreover, the equation cannot be used with triglyceride values >4.5 mmol/L.

In order to obviate these problems, direct assays for LDL are available and have been shown to correlate well with the calculated method.[1] The assays have not been tested in patients with end-stage renal failure, but the structure of LDL is known to be affected by uraemic serum.[2]

This brief study used routine monthly blood samples from 40 diabetic patients receiving maintenance haemodialysis to test the correlation between measured and calculated LDL in uraemia. The results are shown in Figure 1.

The two methods correlated well (r=0.97), but there was a consistent bias, with the 95% confidence interval (CI) falling outside the line of identity. The calculated values were 10–15% lower than the measured values across the measured range (1.0–4.3 mmol/L). The results cannot be explained by hypertriglyceridaemia, as the mean triglyceride value was 1.5 mmol/L (range 0.5–3.8 mmol/L).

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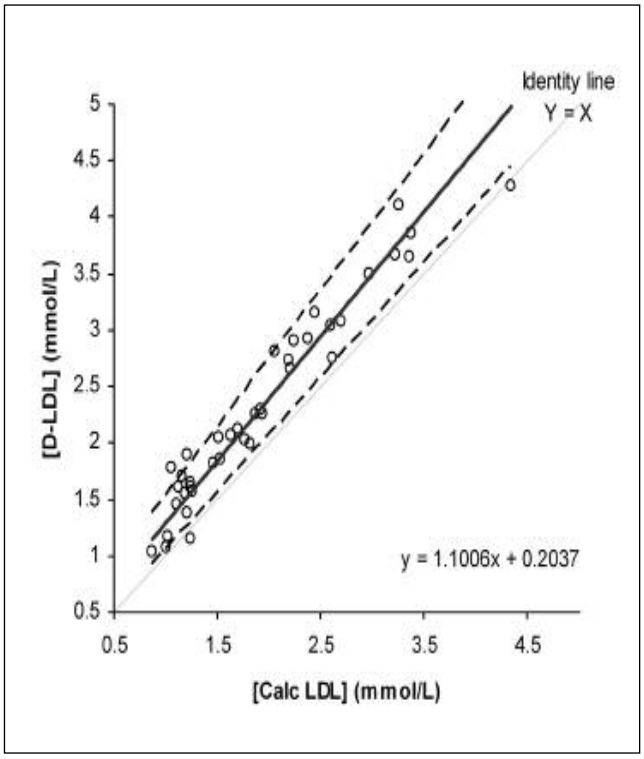

Fig. 1. Correlation between measured and calculated LDL in 40 diabetic patients on maintenance haemodialysis.

Abnormalities of the LDL molecule in uraemia include variations in the proportion of cholesterol and apolipoproteins,[2] and it is possible that these structural changes are responsible for the results presented here. They might also be explained by unidentified retained substances in uraemia interfering with the assay. Whatever the explanation, the discrepancy may have clinical significance as this group of patients has a very high risk for cardiovascular disease.[3]

References

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3 Xue El, Frazier ET *et al*. Association of heart disease with diabetes and hypertension in patients with end-stage renal failure. *Am J Kidney Dis* 2005; **45**: 316–23.

Endocarditis due to a nutritionally variant streptococcus: a lesson in recognition and isolation

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A 40-year-old man with a history of a corrected tetralogy of Fallot was referred by his GP with a six-month history of malaise, lethargy and night sweats. Clinical examination revealed an ejection systolic murmur (grade 4/6) and early diastolic murmur (grade 2/4) in the pulmonary region; however, no old notes were available for comparison with previous examinations. He had a normocytic anaemia (haemoglobin [Hb] 10.7 g/dL, mean cell volume [MCV] 89.5 fL) and a raised C-reactive protein (CRP) of 73 mg/L.

Four sets of blood cultures detected Gram-positive cocci in chains within 24 h (BACTEC 9240 Standard Anaerobic/F and Standard/10 Aerobic/F culture vials, Becton Dickinson, Sparks, MD, USA). A trans-oesophageal echocardiogram showed thickened, hypermobile aortic valve cusps consistent with endocarditis, and the patient was commenced on penicillin and gentamicin.

The blood cultures were subcultured on blood, chocolate, MacConkey and fastidious anaerobic agar (FAA). At 72 h the only growth present was on FAA, with scanty (<0.5 mm

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